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FAT RETENTION IN INFANTS FED BREAST MILK AND HUMANIZED COW'S MILK

PAUL GUILBERT, M.D., DAVID BAKER, M.D., AND L. A. BARNESS, M.D.
PHILADELPHIA, PA.

RECENT investigations of the biological and chemical properties of cow's milk in comparison with those of human milk have led to modification of earlier clinical views concerning requirements for optimal nutrition of infants.[1-6] Additional information is desirable on the mechanisms involved in absorption of nutrients in general and of fat in particular.

Among the known factors that influence fat absorption, the following are well documented: (a) The mineral (alkaline earth) content of the diet,[7a] (b) the percentage of fat present as unsaturated fatty acids,[7a] (c) the length of the carbon chain of the fatty acids,[7a] and (d) the age[7a, 8a] or (e) prematurity[7b, 9, 10, 11, 12a] of the subject. Unless the diet is almost fat-free, the *quantity* of the fat ingested appears to have little effect on absorption. Except in premature infants, the *particle size* of the dietary fat also exerts little influence on fat retention[7a, 12b, 13] in situations when fat is being excreted in the feces in relatively significant amounts. The influence of carbohydrate on fat absorption is still not entirely clear.

Various studies[1, 8a, 14] carried out on full-term, healthy infants have demonstrated that the fat of human breast milk is more efficiently absorbed than the fat of cow's milk.[7, 8, 14] Twenty years ago, Holt's group[7a] found that an average of 92.4 per cent of the fat in a feeding of human milk was absorbed, whereas absorption of fat from a formula derived from cow's milk low in protein with added lactose and vegetable fat was 86.3 per cent. Notwithstanding the fact that the iodine numbers were identical for the two types of feeding, the lower average retention of fat from the cow's milk formula was attributed to a lower content of unsaturated fatty acids. Other possible explanations for the difference in fat retention, as (a) the influence of the fecal pH and (b) the role of intestinal bacteria in the retention of fat from the feeding, have not been suggested.

Thus it will be seen that although several aspects of the subject have been well covered in a number of excellent studies in the past, there is still little information directly correlating the activity of the intestinal flora with

From the Department of Pediatrics, Hospital of the University of Pennsylvania.

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the ability of the host to absorb dietary fat. This is of particular interest inasmuch as the manufacturer of the cow's milk formula studied by Holt and co-workers has, in the years since their investigation, improved the fat mixture and made other alterations in the formula leading to production of a formula in which protein, carbohydrate, and fat content approximate the pattern of human milk. The present study was undertaken to evaluate any possible differences in fat retention, under similar conditions of fecal acidity, which might be detectable between infants fed this formula and those fed human milk.

METHOD

This study was conducted for a total period of one year on five normal male newborn infants.* The age and weight of each infant during each separate observation period are recorded in Table I.

Excluded from the series were infants who gained poorly or were intolerant to milk; those who suffered from gastrointestinal disorders or respiratory infections, and those who previously had had skin or systemic infections or who had received systemic antibiotic therapy for any reason.

Observations were conducted in the nursery under relatively constant conditions of temperature, nursing care, and restriction of activity. A complete physical examination was done on each child before and after the study. Daily records were kept of body weight, volume of water and formula intake, volume of formula refused, number of stools, and volume, pH, and specific gravity of urine.

*The infants were studied at the St. Vincent's Hospital, Philadelphia. Especial note is made of the cooperation of His Excellency, Archbishop John O'Hara, of the Nursing Staff of St. Vincent's Hospital, and of the nurse in charge of the babies, Sister Eustolia.

The diet consisted of human milk alternated with varying dilutions of the modified cow's milk mixture. In four out of five cases the order of feeding was (1) modified cow's milk, (2) breast milk, and (3) modified cow's milk. In one subject, Baby Rus., the sequence was reversed, so that he received (1) breast milk, (2) modified cow's milk, and (3) breast milk. By diluting the cow's milk modification or the breast milk with appropriate amounts of water, lactose and vegetable fat (Lipomul, or olive oil in one case), the diet for each infant was maintained at an equicaloric and equiprotein content approximating 67 calories per 100 ml. The modified cow's milk formula was heated at 80° C. in the Arnold sterilizer for thirty minutes and then terminally sterilized (100° C. for fifteen minutes) just prior to use. The human milk was pooled and kept frozen until ready for use; it was not sterilized.

Feedings were given on a four-hour schedule, ad libitum, and were supplemented by a standard multiple vitamin preparation. Because of the variation in volume of formula ingested daily, the total caloric intake per kilogram of body weight per day was not a constant figure (Table I).

The separate feeding periods were of three days' duration, with carmine used as a fecal indicator for stool collections.

The infants were placed in a Hoag metabolic bed.[15] Stool and urine specimens were collected daily and pooled for each three-day period.

All specimens were frozen until the time of analysis. Stool specimens were treated with concentrated hydrochloride and dried on a steam bath. The dry stool was weighed and ground to a fine powder. An aliquot sample of

TABLE I

SUBJECT	DIET	DURATION OF PERIOD	AGE	WT. (KG.)	TOTAL CAL./ 100 ML. DIET	% OF CAL. FROM FAT	% OF CAL. FROM PROT.	% OF CAL. FROM CHO	CAL./KG./ DAY
F.	SMA	15	21 to 35	3.14–3.41	74	56.5	3.08	40.4	114
	BM	15	39 to 53	3.72	76	55	2.94	42.1	160
	SMA	15	54 to 74	4.17	75.1	57	2.88	40.1	145
Rob.	SMA	15	19 to 33	3.04–3.01	71	50.3	2.76	46.9	131
	BM	15	36 to 50	3.38	78	51.5	2.23	46.3	185
	SMA	9	51 to 69	3.75	80.2	51.6	2.44	46.0	146
Rus.	BM	18	19 to 39	3.88–4.32	67	23.4	6.5	71.0	135
	SMA	27	40 to 68	4.85	64.1	50.9	4.9	44.2	124
	BM	9	69 to 77	5.13	67	39	8.1	52.9	117
S.	SMA	15	12 to 27	3.15–3.39	67.3	55.3	8.15	36.5	139
	BM	15	28 to 42	4.17	67	47	7.29	45.7	157
	SMA	15	43 to 57	4.72	66.5	49.4	12.7	37.9	134
M.	SMA	9	45 to 62	4.17–4.51	65	37.2	9.4	53.4	122
	BM	11	63 to 75	4.89	65	41.7	6.64	51.7	158
	SMA	3	76 to 81	5.03	65	37.2	9.4	53.4	116

TABLE II

SUB.	DIET	DAYS	FAT INTAKE GM./3-DAY PERIOD			STOOL FAT GM./3 DAYS	FAT RETENTION GM./3 DAYS	% FAT RET.	% N RET.	% GRAM + RODS	BIFIDUS	STOOL pH
			FORMULA	LIPOMUL	TOTAL							
F.	SMA	15	29.1	44.6	73.7	9.0	64.7	87	42	75	0	5.9
	BM	15	94.0	65.3	159.3	6.5	152.8	96	60	89	+	5.3
	SMA	15	39.0	70.2	109.2	8.4	100.8	92	53		0	5.4
Rob.	SMA	15	23.2	43.6	66.8	8.2	58.6	88	37	80	0	5.7
	BM	15	22.2	70.6	98.4	8.7	89.7	91	39	92	+	4.8
	SMA	9	26.2	70.3	96.4	4.9	91.5	95	51	93	0	5.3
Rus.	BM	18	43.3	0	43.3	5.5	37.8	87	55		+	5.7
	SMA	27	52.2	43.2	95.4	5.8	89.6	94	55	74	+	5.9
	BM	9	75.9	0	75.9	1.8	74.1	98	65	88	0	5.2
S.	SMA	15	64.8	18.9*	83.7	9.5	74.2	88	59	55	0	5.7
	BM	15	93.0	0	93.1	10.7	82.4	89	58	81	+	5.2
	SMA	15	75.2	22.5	97.7	10.3	87.4	89	49	76	+	5.3
M.	SMA	9	65.6	0	65.6	5.7	59.9	92	47	77	+	5.6
	BM	11	96.9	0	96.9	3.7	92.2	96	51	94	+	5.0
	SMA	3	71.6	0	71.6	10.5	61.1	85	57	74	+	5.4

*Olive oil.

2.0 Gm. was analyzed for total fats by the method of Lewis and Partin.[16] The fat content of the milk formula was determined by a modification of the Roese-Gottlieb method.[17]

The standard error of the mean for these methods is as follows: for stool fat S.E. = ± 1.8 per cent; for milk fat S.E. = ± 2.5 per cent.

Fresh stool specimens were collected every two days for smear and pH determinations. Stools were cultured at least once during the course of each type of diet. Smears were stained with Gram's stain and 200 bacteria were counted and subdivided into gram-positive and gram-negative rods and cocci. The cultures were made on a modification of the Teply and Elvehjem[18] medium and special note was made of the presence of *Lactobacillus bifidus* var. *pennsylvanicus*.

short period of observation—three days, the second period in which infant M. was observed on the cow's milk formula—is excluded from the series, the over-all range for all patients in all periods of observation would be 87 to 98 per cent.

The averages were correlated with the type and order of feeding because of the possible variation of fat retention in relation to age and nature of the previous type of diet. There was a small but definite increase in fat retention as the age of the infant advanced.

The bacterial flora showed a preponderance of gram-positive rods on both types of feedings. Our results were similar in this respect to those of Barbero and associates[19] in a larger group of patients.

TABLE III. AVERAGE VALUES

	SMA			BREAST MILK		
	I	II*	III	I*	II	III*
% fat retention	89	94	90	87	93	98
Bacterial flora % gram-positive rods	72	88	81	--	89	88
Fecal pH	5.7	5.9	5.4	5.7	5.1	5.2

*Observations on Rus. only.

RESULTS

The fat retention after feedings of human breast milk and of the "humanized" cow's milk formula was not significantly different. The results of determinations of fat intake, excretion and retention, stool flora, and pH are recorded in Tables II and III.

The percentage of fat retained from the formula derived from cow's milk varied from 85 per cent (single period of three days) to 95 per cent (total period of nine days). The percentage of fat retention from breast milk varied from 87 per cent (total period of eighteen days) to 98 per cent (total period of nine days). If the one very

L. bifidus var. *pennsylvanicus* was present during the periods on breast feeding except in one instance (Baby Rus.; third period) and also in two other subjects in periods when the diet of modified cow's milk followed a period on breast milk.

The fecal pH ranged from 5.1 to 5.9, and showed no significant difference in relation to the two diets.

DISCUSSION

The role of the *intestinal flora* in nutrition has been the subject of extensive studies[20-22] in recent years, and several investigations have indicated

participation of the bacteria of the large bowel in mediating fat absorption.

Lactose favors production of riboflavin and pyridoxine in the intestine.[23] A high fat diet decreases riboflavin production and development of coliform bacteria. Oleic acid can eliminate the requirement of *Lactobacillus casei* for biotin and is essential to certain lactic acid bacteria, but is toxic for *Lactobacillus bulgaricus*.[19, 20] Yamamoto[24] demonstrated increase of fecal fat in rats and dogs on a protein-free diet, which was explained by either alteration in intestinal flora or diminished pancreatic lipase excretion.

Barbero and his group[19] showed that in infants fed breast milk the stool was distinctly more acid than in infants fed cow's milk preparations. They found also that feeding of a special modification of cow's milk resulted in definitely acid stools, although the intestinal bacteria of the subjects fed this formula differed somewhat from those of the breast-fed infants.

Viswanatha and co-workers[25] studied, in rats, the relation of vitamin E and the carbohydrate (sucrose and lactose) in the diet to the growth-promoting properties of butter fat as compared with those of corn oil. These authors found that a bacteriostatic substance, such as sulfathalidine, could modify the response of the animals to these fats and carbohydrates. Unfortunately, no data on the bacterial composition or pH of the stools were included to throw further light on the role of intestinal bacteria in the retention of fat.

Although it is generally believed that a healthy child can retain adequate fat regardless of the form of fat in a diet, Holt's previous study indicates the fat retention in infants on a formula of humanized cow's milk with low protein, high lactose, and vegetable fat is low. The present study, however, indicates that the fat retention can be increased by modifying the type of fat in the formula toward a more natural combination of vegetable and animal fats. Whether this alteration in fat is responsible for a lower fecal pH or an alteration of intestinal flora is unknown.

SUMMARY

1. Five healthy, newborn male infants were fed diets consisting of "humanized" cow's milk alternated with pooled human breast milk for total periods of nine to twenty-seven days, with an average duration of about fifteen days on each type of feeding.

2. Determinations of fat balance, stool pH, and bacterial flora, including *L. bifidus* var. *pennsylvanicus*, are reported.

3. Contrary to the results of previously reported studies, (a) there was no significant difference in percentage of fat retention on the two types of feedings; (b) the fecal pH and relative numbers of gram-positive rods in the feces were similar for both groups; (c) *L. bifidus* var. *pennsylvanicus* was present in the stools during the periods in which the infants received breast milk and in some periods on "humanized" cow's milk immediately following breast milk feeding. This organism was diminished in periods in which feeding of the modified cow's milk formula preceded breast milk feeding.

Dr. Enid Fischer, Dr. Francisco Torres and Miss Helga Moeksi performed many of the early collections and determinations of this study.

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VITAMIN A DEFICIENCY IN INFANCY

MURRAY H. BASS, M.D.,* AND JOSEPH CAPLAN, M.D.
NEW YORK, N. Y.

VITAMIN A deficiency is unusual in infancy. The average infant fed on milk obtains enough of this vitamin, even without the addition of a vitamin supplement. However, with infants who are allergic to milk and are receiving a milk substitute lacking in animal fats without added vitamin A, the case is different. Such infants may develop a bizarre group of symptoms, baffling to the diagnostician, of serious import to the patient, but one which, if recognized, may be readily and easily cured. The symptoms are so manifold and involve so many organs that the clinical picture should be familiar not only to the pediatrician, but to the allergist, oculist, neurologist, and urologist as well.

CASE REPORT

I. L., the second child of healthy parents, was born at term on Jan. 31, 1954, weighing 7 pounds. At the age of one month he developed a severe, generalized eczema for which his formula was changed from an evaporated milk mixture to a soybean preparation (Mull-Soy). Vitamin C and Drisdol with vitamin A were substituted for the multivitamin preparation he had been receiving. The effect on the eczema was good and he was not seen again by his pediatrician until he was 5 months of age, at which time he appeared well although he weighed only 11¾ pounds. The gradual addition to the diet of cereal, fruit, and vegetable was advised. At 7 months he was again seen for a mild upper respiratory infection with fever of 100° F. One week later blood stains on the diaper led to a urine examination which showed a trace of albumin, many red cells, and some clumped white cells. Achromycin was prescribed but he remained ill and was hospitalized on Oct. 6, 1954.

On admission the baby appeared pale and listless. The temperature was 100.5° F. The skin was normal. The throat was injected. The eardrums were normal. The eyes showed corneal blurring with several small ulcerations. The heart and lungs were normal. The abdomen was soft and the liver and spleen did not appear enlarged. The urine still contained a trace of albumin, some red cells and white cells with a few hyaline casts. The fontanel was flat and there were no abnormal neurological signs.

Blood count showed the following: hemoglobin 6.7 grams, red cells 2,710,000, white cells 18,000 with a polynucleosis of 72 per cent. Nonprotein nitrogen, urea, and blood sugar were normal. Chest x-ray showed normal heart and lungs.

Three small transfusions were given which corrected the anemia. Achromycin was continued but when repeated urine cultures revealed *B. proteus* and *E. coli,* Chloromycetin was prescribed.

On the day following admission, consultation by an oculist revealed: "Ulcerations and infiltrations of the lower portions of the cornea, more marked in the left eye. The location and configuration of the lesions correspond to exposure keratitis, where the interpalpebral portion of the cornea is exposed when the lids remain open. The

From the Pediatric Service of Saint Joseph Hospital, Far Rockaway, N. Y.
*1112 Park Ave.

depth of the anterior chamber is normal. The pupils are normal. The fundi are difficult to visualize because of corneal opacity, but the optic discs appear to be normal. Diagnosis: Corneal ulcers possibly the remnant of exposure keratitis." Hydrocortone eye drops were prescribed and vitamin A by mouth was suggested. At this time it was understood that the baby before coming to the hospital had been continuously receiving vitamin A.

During the next ten days the child's condition became worse. He became more apathetic, took little food, and still ran the low-grade fever. Intravenous pyelogram was done but was not very satisfactory. X-rays of long bones and pelvis were normal. Blood cultures on three different occasions proved sterile.

On September 17 marked bulging of the anterior fontanel was observed. Slight nuchal rigidity appeared and the temperature began to rise, reaching 104.2° F. Lumbar puncture revealed clear, sterile fluid containing sugar 61 mg., chlorides 720 mg., protein 21 mg., and microscopically 3 white cells and an occasional red cell. The fluid rapidly reaccumulated.

A neurosurgical consultant examined the infant and found "moderate irritability; fairly marked nuchal rigidity; distention of the anterior fontanel and scalp veins; seeming limitation in motor power in both lower limbs associated with spasticity and hyperactive reflexes." The diagnoses suggested were subdural hematoma, multiple cerebral septic emboli, thrombosis of the sagittal sinus. Subdural and ventricular taps and repeated fundus examination were suggested. Both subdural and ventricular taps gave negative results. X-ray of the skull was negative.

On September 21 vitamin A deficiency was suggested as the cause of the infant's illness and it was ascertained that instead of Drisdol with vitamin A, the baby, by error, had been receiving plain Drisdol ever since its second month of life. Besides Mull-Soy, his meals consisted of banana, rice, and peaches. In other words he had received no vitamin A either in his food or as a supplement. Antibiotics were discontinued except for a daily injection of penicillin as a prophylactic. Vitamin A, 100,000 units was given daily intramuscularly for six days.

The change in the infant's condition was astonishing. The temperature returned to normal. In two days the fontanel was flat. Within a few days the eyes returned to normal. The baby began to coo and babble, moved about in the bed, and smiled. The urine became normal, and the child's appetite came back.

After six days of intramuscular vitamin A, 25,000 units was given daily by mouth for two weeks, after which an ordinary multivitamin preparation was given. The child was discharged on October 1, ten days after the diagnosis had been made, and has developed well since then. When seen a month later he weighed 14 pounds 5 ounces, a gain of 25 ounces in twenty-eight days. The head measured 16.5 inches, the chest 15.5 inches, and the length 26 inches. The hemoglobin was 12 grams. The urine was normal, and the physical examination was entirely negative. Two months later the baby was reported to be in excellent health, weighing 17.5 pounds.

DISCUSSION

The curious combination of anemia, hematuria, hydrocephalus, and disease of the cornea is so striking that once seen it is not forgotten. In our case the condition was recognized because one of us was familiar with a somewhat similar case published by Cornfeld and Cooke[1] in 1952. These authors describe a 5½-month-old infant, also allergic to cow's milk and kept for several months on a diet consisting of a protein hydrolysate, vegetable fats and Dextri-Maltose (Nutramigen), vitamin D, ascorbic acid, folic acid, and ferrous sulfate. This infant presented signs of

retarded development, hydrocephalus, xerophthalmia, anemia, facial paralysis, and enlargement of the breasts. All of these symptoms could be explained by the absence of vitamin A in the diet and the child made a good recovery when she received large doses of the vitamin.

Blackfan and Wolbach,[2] in an excellent discussion of vitamin A deficiency in infants, reported in 1933 the case of an infant aged 6½ months, weighing 13 pounds 6 ounces, who, having shown signs of allergy to cow's milk, had been kept for several months on a milk substitute (Sobee) and viosterol. Both eyes showed keratomalacia. The neck was retracted and the sutures of the skull were separated. Urinalysis showed numerous white cells with occasional red cells and a large number of epithelial cells. Spinal fluid was normal. The authors say, "Without much doubt vitamin A deficiency disease was initiated as a direct result of an inadequacy of vitamin A in the diet. In addition to the diagnostic sign, keratomalacia, epithelial cells were present in the urine in unusual numbers. The activity of the process in the eye subsided under therapy but with blindness as the resultant effect."

From these cases it is evident that infants kept on a diet lacking animal fats and vitamin A supplement may present an array of symptoms as follows:

1. Retardation of mental and physical growth
2. Anemia with or without hepatosplenomegaly
3. Tendency to infection
4. Epithelial metaplasia
   (a) In the eye: xerophthalmia and keratomalacia
   (b) In the urinary tract: pyuria, hematuria, and excessive numbers of epithelial cells in the urine
   (c) In the genital tract: cornified vaginal epithelium
   (d) In the respiratory and gastrointestinal tracts
5. Hydrocephalus with increased cerebrospinal pressure with or without isolated cranial nerve paralyses
6. Endocrine disturbance: gynecomastia

It has been pointed out by several investigators that these pathological findings in vitamin A deficient infants differ from those seen in adults but closely resemble the findings in experimental animals. Cornfeld and Cooke[1] suggest that this resemblance between the infant and the experimental animal may depend on the fact that they are both rapidly growing organisms.

The vitamin A content of the blood of normal infants varies from 45 to 150 units per milliliter of plasma. "Concentrations below 45 units in infants and below 75 units in children are of significance except in acute febrile disorders and in the newborn period, under which circumstances the low blood levels are transitory and are not associated with poor liver storage."[3] In Cornfeld and Cooke's case the level was 40 U. per 100 c.c. We were unfortunately unable to obtain the level in our case. However, the clinical picture and the extraordinarily prompt disappearance of all the symptoms upon the injection of large amounts of the vitamin leave no doubt as to the diagnosis.

In the case here reported, close questioning of the parents revealed that the baby had been slow in his mental and physical development. At the age of 5 months he weighed only 11½ pounds. For several weeks prior to the child's admission to the hospital he was dull

and listless, and his eyes, as the mother put it, looked "rough." In retrospect, then, we realize that the infant had been showing signs of vitamin A deficiency for some time previous to his acute illness. However, it was the mild upper respiratory infection which ushered in the series of severe symptoms that began with hematuria. As a matter of fact, Clausen[4] states that "the vitamin A level in the blood is lowered in even mild respiratory infections in children." It was probably this loss of vitamin in an organism already depleted that resulted in acute disease.

The presence of frank hematuria following the child's "cold" naturally led to the consideration of acute nephritis or some urinary infection for which he was hospitalized. It is noteworthy that Blackfan and Wolbach[2] also report the case of an infant with vitamin A deficiency admitted to the hospital for "bloody urine." This symptom is the result of the changes in the epithelium of the urinary tract and may be accompanied by the presence of large numbers of epithelial cells in the urinary sediment. In a recently published study of the pathological changes present in acute and in protracted vitamin A deficiency, Stoerk and collaborators,[5] experimenting on rats, found that "in many of the acutely deficient animals, renal tubular changes hitherto not described were noted which perhaps represent another metaplastic change."

The severe anemia exhibited by our patient (hemoglobin 6.7 grams and red cells 2,710,000) is a recognized symptom of vitamin A deficiency.

The ocular symptoms and signs in our case are pathognomonic of vitamin A deficiency. It is usually a late symptom. The conjunctiva looked rough, the cornea appeared cloudy and showed small ulcerations. The frequency of these findings in Danish infants during World War I, at a time when cream and butter were financially beyond the poorer classes, led Bloch to attribute the ocular symptoms to the presence of some substance contained in these animal fats. Between the years 1909 and 1920, 600 to 700 cases of xerophthalmia in children were found to have occurred in a population of less than 3,000,000. When butter was again procurable, xerophthalmia again disappeared from the population. If untreated, this condition goes on to keratomalacia and blindness.

The sudden appearance and later the equally rapid disappearance of increased cerebrospinal pressure with marked bulging of the anterior fontanel and signs of meningeal irritation is of great interest. One cannot reconcile the results of animal experimentation which ascribe this symptom to retardation of skull bone growth with the rapidity of its appearance and disappearance in the human infant. Wolbach and Bessey,[6] as well as Mellanby,[7] point out that in experimental animals the retardation in the growth of the bones of the skull causes pressure on the more rapidly growing nervous tissue. Mellanby says, "It is undoubted that changes of intracranial pressure are produced by vitamin A deficiency; the pressure of the cerebrospinal fluid in the cisterna magna is increased, a condition of slight but definite internal hydrocephalus is often found." Wolbach and Hagsted[8] state categorically that "The neurologic disturbance of vitamin A deficiency in the chick are the result wholly of compression of the central nervous system produced by retardation of growth of

vertebrae and bones of the cranium." In our case the signs of increased intracranial pressure disappeared two days after vitamin A had been administered. This, we believe, shows that to explain such rapid changes in pressure there must be some mechanism at work other than compression due to faulty bone growth.

Increased cerebrospinal pressure has also been reported to occur in calves kept on vitamin A deficient diets, resulting in papilledema, nyctalopia, syncope, and incoordination. When vitamin A was added to the diet these symptoms disappeared.[9]

It is a curious fact that benign hydrocephalus with marked bulging of the fontanel in infants has also been described in *hyper*vitaminosis A.[10, 11] Here it is transient and harmless and disappears rapidly when the intake of vitamin A is reduced. Of this condition Knudson and Rothman[12] say, "The choroid plexus probably produces cerebrospinal fluid at an accelerated rate, another sign of cellular activity."

In this connection the following case recently observed by one of us (M.H.B.) is of considerable interest.*

An infant aged 3 months was proved by operation to have congenital absence of the bile ducts. He was marantic, icteric, with clay-colored stools. There was low-grade fever caused by postoperative intra-abdominal inflammation. During the infant's stay in the hospital it was noted that the anterior fontanel suddenly became very tense and full. There were no neurological symptoms and no increase in the temperature. The ocular fundi showed no abnormal findings. Lumbar puncture revealed clear sterile fluid; microscopically the fluid contained no cells and the chemistry was normal. The fluid did not reaccumulate but the fontanel remained definitely fuller than normal. The vitamin A level in the blood was determined and found very low (34 U. per 100 c.c.). The infant was given daily doses of 50,000 units of vitamin A intramuscularly. After one week the fontanel became flat and remained so until five days later when the child died. At no time were any ocular symptoms present and scrapings of the conjunctiva revealed no abnormality.

It has long been known that hepatic disease with failure to absorb fat leads to vitamin A deficiency, and recently it was shown that the absorption of vitamin A from an oily vehicle parallels the degree of fat absorption as measured by fecal fat balance studies.[13] As long ago as 1924 Wagner[14] reported four children with hepatic disease and marked jaundice who developed keratomalacia. In 1935 Altschule[15] reported on "Vitamin A deficiency in spite of adequate diet in congenital atresia of bile ducts and jaundice." At autopsy microscopic evidence of vitamin A deficiency was found in six of eleven infants studied. He says, "The presence of the deficiency may be overlooked because the specific changes of vitamin A deficiency occur late in the course of the disease."

It is suggested that the sudden variations in the cerebrospinal fluid pressure in our second case may well have been due to vitamin A deficiency.

SUMMARY

The clinical picture of vitamin A deficiency in infants is discussed and two cases are reported. An allergic infant kept for five months on a diet which contained no milk, no animal fats, and no vitamin A supplement developed malnutrition, severe anemia, hydrocephalus, hematuria, and xeroph-

*This patient was seen at the North Shore Hospital, Manhasset, L. I., through the courtesy of Dr. Alfred Florman.

thalmia. All these symptoms promptly responded to large doses of vitamin A and the child made a complete recovery.

The second infant, deeply icteric due to proved congenital absence of the bile ducts, with a low vitamin A blood level, developed evidence of acute hydrocephalus which disappeared with intramuscular administration of vitamin A. The possibility of this symptom being due to the low vitamin A level is suggested.

CONCLUSION

Vitamin A deficiency in infancy may cause many varied symptoms and may lead to very serious disease. Our first case is particularly important as a reminder and a warning to pediatricians that when allergic infants are put on a milk-free diet, the addition of vitamin A to the diet must not be overlooked.

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MOTORIST INJURIES TO CHILDREN

JACOB KULOWSKI, M.D.
ST. JOSEPH, MO.

IN A series of 661 motorist casualty survivors, as distinguished from all other traffic accidents, there were 45 (approximately 7 per cent) patients under 15 years of age.* Table I establishes the age and sex distribution among these children. The sexes were evenly divided. There were 5 infants, 3 preschool, 16 between 5 and 10 years, and 21 children between 10 and 15 years, respectively. Table II shows the slight rise in incidence of motorist injuries between the ages of 15 and 16 years, and the sharp rise which occurs in the age group between 16 and 20 years. The implications with regard to driver proficiency and passenger exposure are obvious, considering the fact that the latter group made up 86 per cent of all patients in the second decade in the entire series of 661 cases. In the age group between 15 and 16 years there were six drivers (27 per cent), 41 per cent front seat passengers, 14 per cent rear seat passengers, and 18 per cent who could not be classified in regard to their seating.

In the age group between 16 and 20 years the seating was as follows: drivers, 29 (35 per cent; males 21, females 8); front seat passengers, 21 (26 per cent; males 10, females 11); rear seat passengers, 10 (12 per cent; males 5, females 5); and unclassified occupants, 22 (27 per cent; males 13, females 9), respectively. There were 82 persons in this category, 49 males and 33 females.

Table III lists comparatively the incidence of the various bodily areas injured and the character of the lesions, whether soft tissue surface or skeletal ones. The frequency rates for these bodily areas for the series as a whole (661 cases) were in the following order: extremity, head, face, chest, trunk, neck, pelvis, and abdomen. For our children, this is face, head, extremity, neck, pelvis, chest, abdomen, and trunk. Except for the head, there is a relative increase in skeletal injury among the older age group. Moreover, there were no serious internal injuries among the children, whereas there was brain injury in 3 persons and a ruptured kidney in 1 person among the older age group. Shock was prominent in only 2 of the children (about 4 per cent) and in 4 (approximately the same per cent) of the older age group. Table IV establishes the nature of the lesions among the children in detail with regard to fractures. In addition to the fractures there were 21 with lacerations, 20 with contusions, and 13 with abrasions. There were relatively few strains, sprains, and dislocations.

In those under 2 years there was only one fracture, that of the skull. The remainder had only soft-tissue injuries. In the age group from 2 to

Address: 413-17 Corby Bldg.

*The entire series of cases consisted of patients admitted to the Missouri Methodist Hospital from late 1949 through 1954, and were treated by various members of the medical and surgical staffs.

5 years, the three patients exhibited one fracture of the femoral shaft, a fracture of the nose, and bodily contusions, respectively. The 15 patients between 5 and 10 years received 12 fractures in all, a high percentage. Twenty-one of the last group (10 to 15 years) exhibited 14 fractures.

COMMENT

This small series of pediatric motorist injuries considered on the background of the more numerous and various other accidents that children experience might seem relatively unimportant. The same kind of an impression unfortunately derives also

TABLE I. AGE AND SEX DISTRIBUTION AMONG 45 CHILDREN MOTORIST SURVIVORS

AGE IN YEARS	MALE	FEMALE	TOTAL	
			NUMBER	PER CENT
Up to 2	1	4	5	11.0
2 to 5	2	1	3	7.0
5 to 10	9	7	16	36.0
10 to 15	10	11	21	46.0
Total	22	23	45	100.0

TABLE II. AGE AND SEX DISTRIBUTION AMONG 149 MOTORIST SURVIVORS OF THE FIRST TWO DECADES

AGE IN YEARS	MALE	FEMALE	TOTAL	
			NUMBER	PER CENT
Up to 15	22	23	45	30.0
15 to 16	14	8	22	15.0
16 to 20	49	33	82	55.0
Total	85	64	149	100.0

TABLE III. FREQUENCY OF BODILY AREAS INJURED AND CHARACTER OF LESIONS*

BODILY AREA	UP TO 15 YEARS		15 TO 20 YEARS	
	SURFACE	SKELETAL	SURFACE	SKELETAL
Head	14	4†	37	2‡
Face	21	9	34	18
Extremity§	6	10	35	31
Multiple¶	6	0	10	0
Neck	3	1	1	8
Chest	2	1	12	6
Abdomen	3	0	1	0
Trunk	1	1	7	6
Pelvis	3	1	1	4
Total	59	27	138	75

*Calculated on basis of areas affected; not on the number of lesions in each area.
†Associated with eight cerebral concussions.
‡Associated with twenty cerebral concussions.
§Practically all fractures but include a few dislocations.
¶Refers to multiple bodily contusions, lacerations and/or abrasions as to the lesions designated under "surface."

TABLE IV. FREQUENCY AND CHARACTER OF INJURIES AMONG 45 CHILDREN SURVIVORS

Head 4	Face: maxilla 4, nose 3, mandible 1, zygoma 2, teeth 2
Femur 3 simple; 2 compound	Radius 2
Tibia 1	Ulna 2
Fibula 1	Humerus 1
Metatarsal 2	Hand 1
Spine 1	Ilium 1
	Sternum 1

from any evaluation based on survivors alone. Survivors always tend to exaggerate the remarkable tolerance of the human body to mechanical injuries. A different picture results from even a brief consideration of 3 fatal motorist casualties in children among 26 autopsy records studied (12 per cent).

CASE 1.—Sharon T., female, aged 3 years, died seven hours after admission to the Missouri Methodist hospital. She was in the back seat with several other sisters when the car crashed into the back of a stalled truck one dark and rainy night. All told, the nine people in that car were hurt in varying degree, proving that packaging for safety does not mean overcrowding. Autopsy revealed only focal areas of cerebral petechial hemorrhages, *without skull fracture*. She had died in hyperpyrexia. Already, however, there was evident passive congestion of the lungs.

CASE 2.—Dorothy P., aged 11 years, died eight hours after admission to the University of Kansas Medical Center. She was forcibly ejected from the rear of a pick-up truck and struck her head on the pavement. Autopsy revealed separation of the suture lines of the skull at the frontal, parietal, and temporal areas. There was multiple and focal hemorrhages in the midbrain, subarachnoid hemorrhage in the brain stem, and moderate edema and blood in the ventricles. She received a severe *contusion to the heart* as evidenced by advanced subendocardial hemorrhage in the left ventricle. The left optic nerve had been severed. There were focal hemorrhages in the spleen, and an advanced bronchopneumonia.

CASE 3.—Patricia M., aged 3 years, died three hours after admission to the University of Kansas Medical Center, after having been forcibly ejected on collision impact and run over. The right leg was practically amputated, the left tibia and fibula also fractured. The spleen was ruptured, the right lung was lacerated and associated with a hemothorax, the brain was contused (*without skull fracture*) and showed subarachnoid hemorrhages and edema with compression, and terminal hyperpyrexia. Among the complications worthy of mention were a lower nephron nephrosis, secondary shock (marked congestion of all the viscera), fatty liver, fat embolism of the lungs, and a marked tracheobronchitis with considerable exudate in the bronchi.

Here, then, concentrated into three small bodies are all of the most deadly complications with regard to motorist casualties: severe injuries of the extremities, multiplicity of injuries with emphasis upon internal ones of the head and those above and below the diaphragm, either alone or in combination. Their challenges are to be met in chronological order in so far as it is humanly possible to do so.

For example, first aid for serious injuries of the extremities is a must, but has practically disappeared from the traffic accident scene. The need, here, is for revised and simpler methods of first aid. Pediatricians can sponsor this movement first through their young groups in scouting, etc., but the basic responsibility ought to be with Red Cross, Civil Defense, and the medical profession as a whole.

For the vast majority of victims (children as well as others) who survive for a day or two and then succumb, the need is for intensive emergency care, with special attention to shock (from hemorrhage chiefly), pulmonary ventilation (oxygen, suction, and aspiration and tracheotomy), and alertness for cardiac arrest. Regarding definitive treatment the emphasis should be alertness to such complications as fat embolism, urinary output alterations, pulmonary conditions, and further alertness to more subtle diagnostic problems offered by internal in-

juries of the major bodily cavities. Out of this program the survival rate will go up and with it will ensue further challenges to therapy for all branches of medical and surgical fields of endeavor as yet untried. For the present it may be necessary to change our policies to some extent with regard to considerations of poor surgical risks, and allow the exigencies of the individual case, backed by competent authority, to decide the issues of therapeutic priorities as well as surgical interventions.

From the standpoint of mortality differentials (intermediate and delayed deaths having been discussed by inference already) there remains the immediate mortality during collision impacts. This probably comprises 10 to 20 per cent of all motorist casualty fatalities. These results epitomize the responsibilities of engineers and manufacturers since there are no other contributory causes of death operative in this small but highly lethal group.

CONCLUSIONS

The basic responsibility of medical groups remains the active and specific treatment of any and all kinds of motorist injuries, always giving priorities to the seriously injured.

The common denominator here is force, the magnitude of which may be less important to crash victims than its source. Epidemiological methods of study demand therefore, among other things, that the sources of these mechanical injuries be scientifically evaluated. It is being learned that patterns of injury emerge from certain collision impacts and/or upsets, despite the fact that basically the interactions between anatomy and structure under crash conditions is one of human versus mechanical variables.

Be that as it may, pediatricians, because they have been interested in preventive medicine over a broad spectrum of situations, are in a position to help keep alive current interests in possible prophylactic measures regarding motorist injuries, both from human and crash impact engineering points of view.

However, the pediatrician can do even better in promulgating accident prevention in the first place through, first, educating parents as to their responsibilities to their occupant children, and, second, by encouraging any and all kinds of driver-training programs at his own community level, remembering that soon the child passenger occupant will become the driver.

A STUDY OF QUINIDINE DOSAGE IN INFANTS AND CHILDREN

GWEN NEWTON, M.D., AND ELIZABETH J. CRAWFORD, B.S.
ST. LOUIS, MO.

CARDIAC arrhythmias such as auricular fibrillation and paroxysmal auricular tachycardia are infrequently seen in infants and children. The digitalis group of compounds has been the treatment of choice for these disorders and in most instances has been found to be effective. There are relatively few reports about the use of quinidine in children with cardiac arrhythmias.[14] It may well be that there is reluctance to try the drug because little information is available on absorption, cardiac effects, dosage, and toxicity of quinidine in infants and children. As such knowledge becomes available, quinidine may also be found particularly useful during cardiac catheterization procedures and in treatment of patients undergoing surgical repair of interauricular defects.[7]

This study was undertaken in an effort to establish a tentative dosage schedule for administration of quinidine in children, based on determination of serum levels of the drug. Included in the paper will be the experimental basis for the dosage schedule, together with the results of application of the proposed schedule in normal infants and in a case of a child with impure auricular flutter. The method used in determining quinidine content of the serum will be presented in some detail.

In conjunction with research on antimalarial drugs during World War II several methods for determining quinidine content of body fluids were developed.[2-5] Since that time various investigators have determined blood quinidine levels as related to dosage, cardiac effects, and toxicity in adults. And, since most observers agree that blood quinidine levels tend to parallel cardiac effects of the drug,[8, 13, 16] it seemed practical to approach the problem of quinidine dosage in children by utilization of blood levels.

The study may be conveniently divided into two sections: the first concerns observations made following single doses of quinidine, orally and intramuscularly, while the second section relates to an evaluation of two different schedules for multiple doses.

The case material consisted of infants ranging in age from a few days to 2 years. All were considered to have normal hearts by physical examination and history. In addition, base-line electrocardiograms were taken on all children scheduled to receive multiple doses.

Preparations for oral use included quinidine sulfate in powder form and a liquid preparation of quinidine sulfate in an alcoholic vehicle, which was mixed with honey for administration. For the intramuscular route three preparations were used: quinidine sulfate in propylene glycol, quinidine hydrochloride with 15 per cent antipyrine and 20 per cent urea, and quinidine gluconate.

From the Departments of Pediatrics and of Pharmacology, Washington University Medical School.

The following procedure was used. The dosage for each child was calculated, using two methods: either according to body weight or surface area. We attempted to arrange a time of administration which would fall between feedings, since at least one observer has noted lower blood levels when quinidine is given immediately after a meal.[11] Following administration of the drug, capillary blood samples were collected at measured intervals. The serum was separated from the cells, and was stored under refrigeration for periods ranging from a few hours to a week before analysis was carried out. (Various observers have noted stability of quinidine samples stored for periods of two weeks or more.[9]) All serum samples were analyzed for quinidine content, using a direct fluorometric method.

METHOD OF ANALYSIS

A method involving the direct reading of serum fluorescence in sulfuric acid, which requires only 0.05 ml. of serum, was used for all samples. This method measures quinidine plus any similarly fluorescing quinidine metabolites.

To determine fluorescence a Farrand-type microfluorometer was used, but any fluorometer would be satisfactory, provided the sensitivity is sufficient to give a full-scale reading with 0.05 mg. quinidine per liter, and the optical system is adequate to give a reasonably low blank reading at this sensitivity. The "B_1" filters (primary Corning 5860, secondary 4308 and 3389) were used.

Reagents.—

0.1N H_2SO_4
5 M $CaCl_2$
Standard (quinidine sulfate, 5 mg. per liter of H_2O)

Procedure.—

Place 0.05 ml. of serum (measured with a Levy-Lang type constriction pipette) in an 18 by 150 mm. fluorometer tube. Add 7.0 ml. of sulfuric acid. Mix well without inverting and read immediately = R_1.

Add 0.05 ml. of the quinidine standard, mix, and read immediately = R_2.

To quench fluorescence due to quinidine, add 0.2 ml. of calcium chloride, and read = R_3.

$$\text{Mg. quinidine sulfate per liter} = \frac{R_1 - R_3}{R_2 - R_1} \times \frac{\text{volume of standard}}{\text{volume of serum}} \times 5 \text{ mg. per liter}$$

We have ignored the slight dilution involved, which results in values about 0.7 per cent too low. By using the same pipette for both serum and internal standard the pipette correction cancels out.

Serum itself contains enough chlorides to cause some quenching of quinidine even at this 1/140 dilution. Hemolysis may also cause a slight decrease in fluorescence. It is for these reasons that an internal standard which reads one to two times the initial fluorescence is used. Immediate reading minimizes possible slow drop in fluorescence due to glass binding of quinidine at these low concentrations.

In an effort to evaluate the accuracy of the direct method, duplicate determinations were carried out. In the course of the study, over 225 quinidine levels were determined; about a third of these were run in duplicate. The difference was no greater than 0.3 mg. per liter in any two readings of a single sample.

In order to compare our results (using the direct method of quinidine determination) with values reported in the literature (many of which are based on extraction procedures), a series of fifty-four samples was simultaneously analyzed by the direct method and by an adaptation of Brodie and Udenfriend's double extraction

method.* Brodie had found that quinidine is completely extracted from an alkaline solution by benzene; only a small amount of the fluorescing quinidine metabolites is carried over.[3, 5]

Results of simultaneous determinations revealed that, in general, levels by the direct method were twice as high as levels obtained by extraction. If the difference, which is equivalent to metabolites, is expressed as percentage of the total reading (by direct method), an average of 47.5 per cent metabolites was noted two hours after a single oral dose, with a range of 39 to 64.5 per cent. With multiple doses the percentage of metabolites seemed to level off at values of 60 to 65. Determinations of percentage of metabolites following single intramuscular doses roughly paralleled the findings with oral doses. These values are compiled in Table I.

TABLE I. AMOUNT OF QUINIDINE METABOLITES IN SERUM AS MEASURED BY DIRECT METHOD

Single Doses

ROUTE OF ADMINISTRATION	ORAL	INTRAMUSCULAR	
TIME AFTER DOSE	1½ – 2½ HRS.	1 – 1¼ HRS.	2 HRS.
% Metabolites	48.5 41.0 42.0 39.0 54.0 43.0 64.5	50 70 35 32	57.0 76.5 44.5 40.0
Average	47.5 %	47%	54%

$$\frac{\text{Direct method—Extraction method}}{\text{Direct method}} = \% \text{ Metabolites}$$

Multiple Doses (Oral)

DOSAGE SCHEDULE	EVERY 2 HR.	EVERY 4 HR.		
DOSE	FIFTH	FOURTH	FIFTH	SEVENTH
% Metabolites	68 57 52	74 63 64 50	63 67	50 70
Average	59%	63%	65%	60%

*Procedure for extraction method: 150 c.mm. of serum, 150 c.mm. of 0.1N NaOH and 500 c.mm. of benzene are mixed (in a 10 by 75 mm. test tube) by buzzing against a rapidly rotating eccentric rod for about 15 seconds. (Too vigorous mixing may result in emulsions.) The tubes, capped with aluminum foil, are centrifuged cold. Next, 17 c.mm. of isoamyl alcohol is added to the benzene layer and mixed without disturbing the aqueous layer. 270 c.mm. of this benzene-isoamyl layer is transferred to an 18 by 150 mm. calibrated fluorometer tube containing 7 ml. of 0.1N H_2SO_4, mixed by shaking without inverting, and allowed to sit until clear (about three minutes). The fluorescence is then read, and the quinidine content calculated by comparing with known aqueous quinidine standards simultaneously run.

RESULTS

The results of preliminary studies, the method for setting up a multiple dosage schedule, and the results of a trial of this schedule are described in the following paragraphs.

In the preliminary study fifty-four children were given single doses of quinidine: thirty of these were given oral quinidine with eighty-seven subsequent serum levels; twenty-four children received intramuscular quinidine with sixty-two subsequent levels. They may be divided into three groups:

Group 1.—A total of fourteen children received oral quinidine sulfate, 5 mg. per kilogram of body weight. The maximum quinidine serum level, which was reached by two hours, averaged (thirteen cases) 1.7 mg. per liter. At four hours the average level was 0.96 mg. per liter, while there was a still measurable level at eight hours in all cases.

Group 2.—A total of sixteen children were given oral quinidine in dosages of either 10 mg. per kilogram, or 200 mg. per square meter of surface area. The maximum level was reached by two hours in all but two cases. Three determinations were made at forty minutes; of these, one represented a maximum value, while the other two reached a maximum by one and one-fourth hours. The average maximum level was 4.6 mg. per liter, with a range of 1.9 to 7.2 mg. per liter. Levels at various times, together with the standard deviation, are tabulated below:

2-hour level—4.1 mg./L. (av. of 13 cases) S. D. ± 1.4.
3-hour level—3.5 mg./L. (av. of 14 cases) S. D. ± 1.4.
4-hour level—3.0 mg./L. (av. of 8 cases) S. D. ± 1.8.

Thus, while the maximum level was reached in two hours, the curve tended to fall off slowly during the ensuing two hours. This may be seen in Fig. 1.

Group 3.—A total of twenty-four children received intramuscular quinidine in dosages of 5 mg. per kilogram of body weight, or 100 mg. per square meter of surface area. An attempt was made to evaluate the relative advantages of calculating dosage by surface area as opposed to body weight, but efforts to obtain more uniform levels with dosage calculated by surface area were inconclusive.

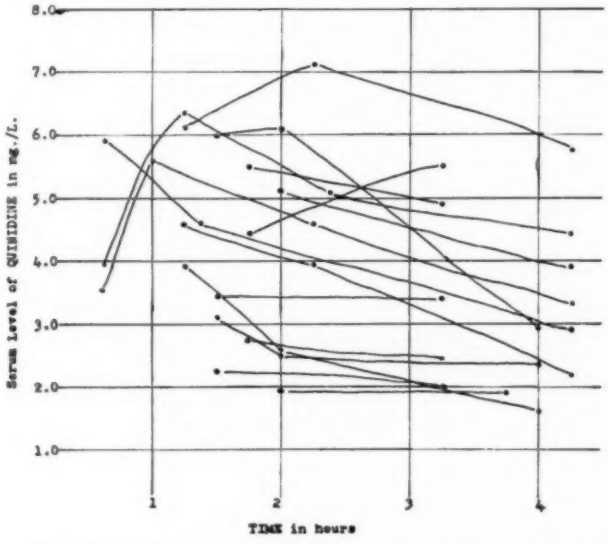


Fig. 1.—Single doses of oral quinidine (10 mg. per kilogram of body weight).

Because the range of levels within the two groups was about the same, the groups were combined in calculating the average levels obtained after intramuscular quinidine. Blood samples taken between one and one and one-fourth hours after administration of the drug represented maximum values obtained in fourteen cases. Average values obtained at various times are tabulated below with the standard deviations; curves obtained are shown in Fig. 2.

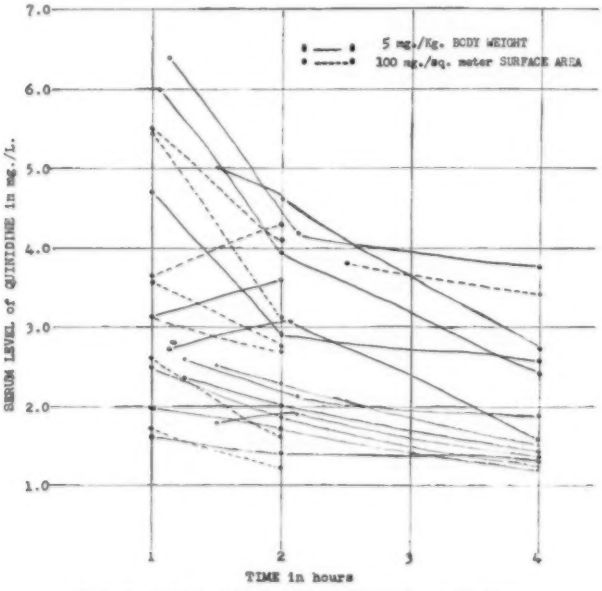


Fig. 2.—Single doses of intramuscular quinidine.

1-hour level—3.8 mg./L. (av. of 12 cases) S. D. ± 1.6.
1½-hour level—3.1 mg./L. (av. of 10 cases) S. D. ± 1.5.
2-hour level—2.8 mg./L. (av. of 21 cases) S. D. ± 1.2.
4-hour level—1.9 mg./L. (av. of 12 cases) S. D. ± 1.0.

A comparison of various intramuscular quinidine preparations was made concomitantly: twelve children received quinidine gluconate, seven received quinidine hydrochloride, and five received quinidine sulfate. Resulting levels showed no variations which could not be explained on the basis of individual differences.

Results of preliminary studies, comparing oral and intramuscular quinidine, suggested that with equal dosage higher serum levels would be obtained with intramuscular preparations, the maximum level would be reached more quickly, and the serum level would decline more rapidly.

In approaching the problem of a suitable schedule for multiple dosage, we decided to utilize the concept of half-time, hoping we might in this way eliminate some of the guesswork and obtain predictable blood levels.

Half-time for a drug is perhaps best defined as the time required for any single dose of the drug to be half dissipated. If blood levels of a drug are

determined at intervals after a given dose, the rate of fall from the maximum level follows a curve, and the logarithmic relationships of the slope of such a curve are used to calculate the half-time. Thus, we arrived at a value of four hours for the half-time of oral quinidine in infants. By way of comparison, in adults a single dose of quinidine is half dissipated in eight hours.[17]

There are two convenient "rules of thumb" which may be applied, once half-time is known, to calculate rate of cumulation and the maximum blood level when rate of cumulation is equal to rate of dissipation of the drug, i.e., the plateau value. The rule for determining rate of cumulation says that a blood level approaches 50 per cent of the maximum level to be reached in one half-time (in our case, four hours); 75 per cent of the maximum is reached in two half-times, 87 per cent in three half-times, and so on. Thus, by 16 hours (four half-times), the serum level would be 93 per cent of the maximum level and for all practical purposes a plateau would have been reached.

The rule for determining the maximum blood level to be reached requires that both half-time and an average serum level after a given dose of a drug be known. This rule may best be expressed by a formula:

$$\text{No. of times a drug is given in 1.5 half-times}^{*} \times \frac{\text{serum level}}{\text{p. single dose}} = \text{maximum level to be attained}$$

*The mathematical basis for the derivation of the value of 1.5 can be found in any standard mathematics text. Briefly, the value is derived from the formula for geometric progression starting with an expression for the value of the residue for any single dose:

$$\text{Residue} = 0.5^{\frac{\text{Total time elapsed}}{\text{Half-time of drug}}}$$

Thus, if 10 mg./kg. of quinidine gives a serum level of 4 mg./L.
if half-time of oral quinidine is 4 hours
if the above dose is given q. 2 hr.
then, the maximum level can be calculated as follows:

1.5 × half-time of 4 hr. = 6 hr.
No. of times drug given in 6 hr. = 3
3× 4 = 12 mg./L.

We arbitrarily decided to aim for a serum level of 9 mg. per liter. By using simple proportions we theorized that a 7.5 mg. per kilogram dose should give a two-hour serum level of 3.0 mg. per liter. By calculation, such a dose given every two hours should result in a maximum serum level of 9 mg. per liter. If we doubled the dose, but gave the drug on a four-hour schedule, we should arrive at the same maximum serum level. At the end of one half-time (four hours) the level should be 50 per cent of the maximum, or 4.5 mg. per liter; the eight-hour level should be 6.7 mg. per liter; and a plateau should be reached by 16 hours.*

To evaluate the tentative dosage schedule fourteen children were given oral quinidine on a two-hour or four-hour schedule, with a total of seventy-three subsequent blood levels. Thus, with a dosage schedule of 15 mg. per kilogram every four hours, four children had seven or more doses with levels taken two hours after a dose. While each child showed a fluctuation of about 3 mg. in levels subsequent to the third dose, there is little indication that levels were continuing to rise, and it seems probable that a plateau was reached at about 12 hours. In general, the levels ranged slightly higher than the predicted maximum of 9 mg. per

*Strictly speaking, since the 2-hour level after a single oral dose is considered the 100 per cent level, zero time should be two hours after the first dose is given. Since the difference in expected levels is relatively small, we have, for convenience, considered the time of administration of the first dose as zero-time.

liter. The curves obtained with these four children and the theoretical curve are plotted on the graph in Fig. 3.

It is of interest to compare these findings with reports on adults given multiple doses of quinidine, since various observers[16, 19] have noted that a plateau is reached in two or three days.

It has also been noted, in adults, that a dosage plan of five doses given two hours apart will provide a residual morning level of some 40 per cent of the previous maximum.[17] With the shorter half-time in children, it did not seem likely that such a plan would be practical; by calculation, such a plan

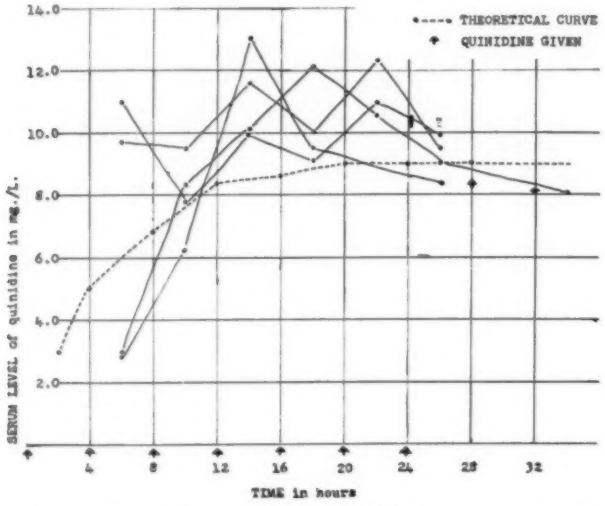


Fig. 3.—Multiple doses of quinidine (15 mg. per kilogram orally every four hours).

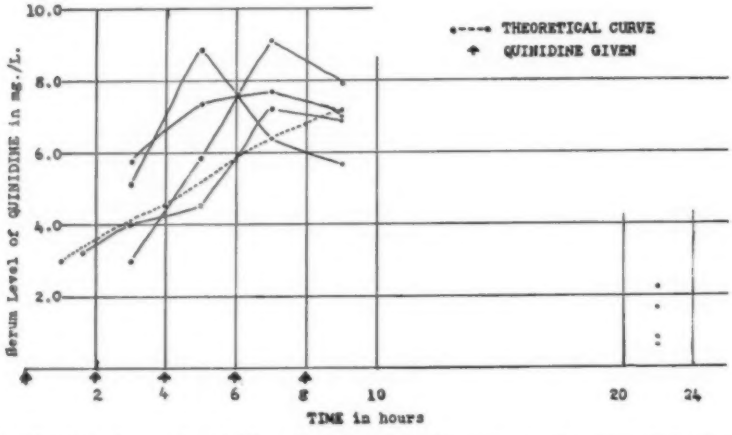


Fig. 4.—Multiple doses of quinidine (7.5 mg. per kilogram orally every two hours, five times).

would provide a residual of only 12 per cent of maximum level in an infant. In an effort to demonstrate this, six children were placed on a two-hour schedule of 7.5 mg. per kilogram, which by calculation should give a serum level of about 7.2 mg. per liter after five doses, and a residual level (12 hours after the last dose) of 0.9 mg. per liter. One child was carried through four doses, while three children received five doses. As can be seen in Fig. 4, levels obtained again showed fluctuation but were close to the predicted level when the drug was stopped. Residual levels were in the predicted range of 12 per cent of maximum in two cases, while one child had a residual of 22 per cent and one had a residual of 35 per cent. (If half-time begins to approach that of the adult as the child grows older, then conceivably the child who had the residual of 35 per cent might have a longer half-time, since he was 18 months old, in contrast to the other three, who were less than 6 months old.)

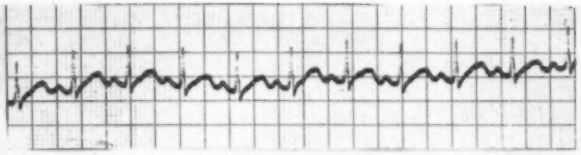
Children receiving multiple doses were followed by electrocardiograms, which were compared with base-line studies for any evidence of quinidine effect. According to various observers[10, 15] the most consistent findings of quinidine effect by electrocardiogram are flattening, broadening (and occasional notching) of T waves, and an increase in duration of the Q-T interval.

A review of the thirty-three electrocardiograms taken in the course of the study makes two facts immediately evident: the only electrocardiographic change seen among the normal children receiving quinidine was a qualitative change in the appearance of the T wave; there were marked individual differences with little evidence of correlation between blood levels and cardiac effects of the drug, as determined by electrocardiograms.

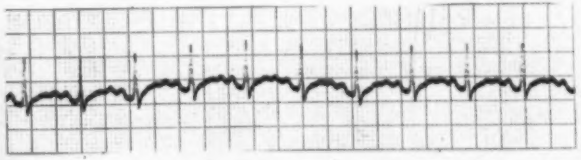
In summary, there was no apparent increase in duration of the Q-T interval; individual differences ranged from no change to slight flattening of the T waves to appearance of a "U" wave in one case. In two cases evidence of quinidine effect was noted within six hours after the drug was started, but subsequent electrocardiograms were identical with base-line studies in spite of continued administration of the drug. Examples of the various electrocardiographic findings are shown in Fig. 5.

Because many of the untoward effects ascribed to quinidine are subjective symptoms labeled cinchonism, which obviously cannot be evaluated in the infant, we relied on observations of vomiting, diarrhea, and fever as evidence of intolerance to quinidine. There was one case in which oral quinidine appeared to provoke vomiting; no other evidence of intolerance was noted among the normal children.

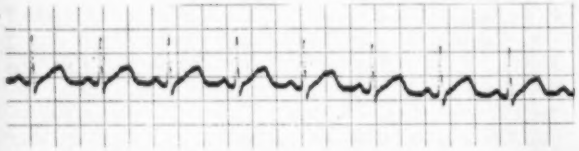
During the course of the present study we had an opportunity to evaluate the use of a tentative dosage schedule of quinidine in a child with cardiac arrhythmia. Because the patient (who was 4½ years old) was older than the age group tested in the study, we reasoned that dose requirements would be smaller. We arbitrarily selected 5 mg. per kilogram as an initial dose, and found that we were able to adjust the dosage to a satisfactory value with a minimum of serum level determinations.



A. Baseline

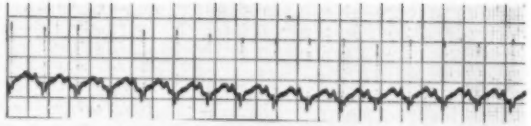


B. T waves lower in amplitude. "U" wave present. Quinidine level of 7.4 mg./L. after 3rd dose (q 2 hrs.)

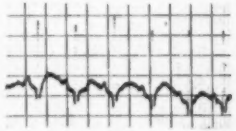


C. Return to baseline after discontinuing drug.

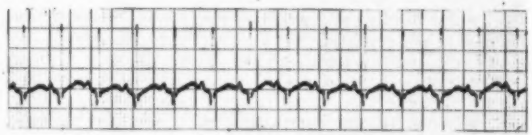
1.



A. Baseline. Sinus tachycardia.



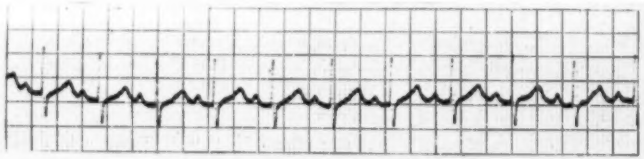
B. T waves broad. Quinidine level of 4.4 mg./L.



C. T waves broad and slightly lower in amplitude. Quinidine level of 7 mg./L.

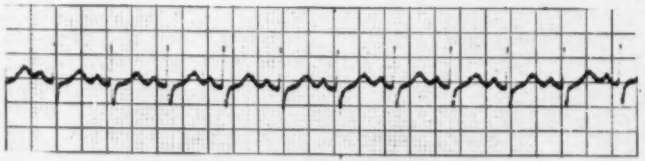
D. Return to baseline after discontinuing drug.

2.



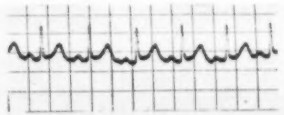
A. Baseline

B. T waves slightly flattened. Quinidine level of 9.4 mg./L.

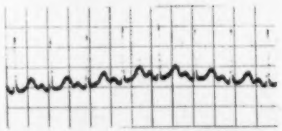


C. Return to baseline while on the same dosage. Level 10 mg./L.

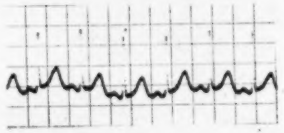
3.



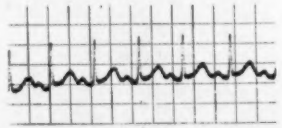
A. Baseline



B. Slight decrease in amplitude of T. waves. Quinidine level of 8.6 mg./L.



C. Return to baseline while on the same dosage. Quinidine level of 8.7 mg./L.



D. No change with quinidine level of 20 mg./L.

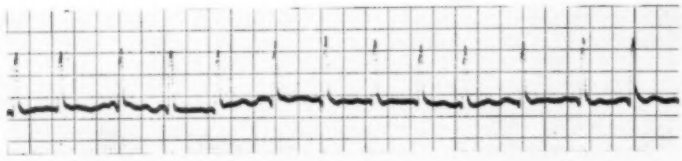
4.

Fig. 5.—Electrocardiographic tracings during quinidine administration to normals. *1*, Patient C. C., showing quinidine effect. *2*, Patient J. E., showing quinidine effect with low levels. *3*, Patient J. B., showing return to base line on continued dosage. *4*, Patient T. J., showing lack of correlation between quinidine level and cardiac effect.

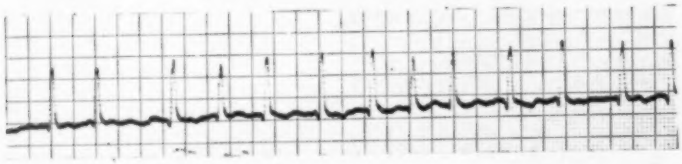
CASE REPORT

A 4½-year-old girl with known congenital heart disease was admitted to St. Louis Children's Hospital on Dec. 29, 1954, with fever, left lower lobe consolidation on x-ray, early cardiac failure, and impure auricular flutter by electrocardiography (Fig. 6).

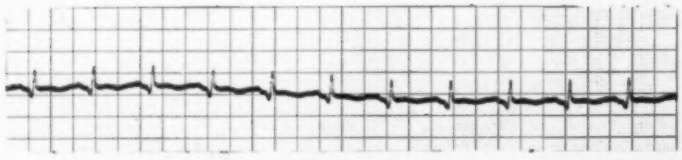
The child had been followed in outpatient clinic for more than one year and had had two previous hospitalizations. Her cardiac anomalies were incompletely known, although left ventricular enlargement was noted on electrocardiogram, generalized cardiac enlargement could be seen on x-ray, right ventricular hypertension had been found in cardiac catheterization, and angiograms showed no filling of the left pulmonary artery.



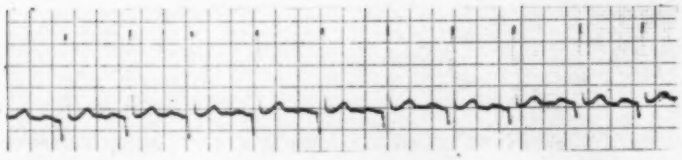
A. Before quinidine. Impure flutter. Lead 2.



B. No change. Quinidine level of 5.4 mg./L. Lead 2.



C. Sinus rhythm. Quinidine level of 10.5 mg./L. Lead 1.



D. Sinus rhythm maintained without quinidine. Lead. 2.

Fig. 6.—Response of auricular flutter during quinidine therapy. Patient L. M.

Cardiac decompensation had occurred for the first time one year prior to present admission; the child had responded well to digitoxin. A second episode of failure seven months before the present admission had again responded to digitoxin, and she had been placed on maintenance dosage.

Repeated electrocardiograms showed a persistence of the impure flutter rhythm, and since the possibility of digitalis intoxication existed, digitoxin was stopped one week after admission and potassium chloride was begun. Because the irregular rhythm was noted to persist, redigitalization was begun eight days later. This was followed by double doses of maintenance digitoxin, to be sure the child was fully digitalized.

The impure flutter rhythm continued, and the decision was made to try quinidine; accordingly, digitoxin was stopped. Six days later on Jan. 28, 1955, oral quinidine was begun on a dosage schedule of 5 mg. per kilogram (70 mg.) every two hours. Serum levels after the second and third doses were 4.7 and 5.4 mg. per liter, respectively. We had elected to aim for a level of 10 mg. per liter; while we could not be sure of the exact half-time of the drug in this child, the levels obtained after three doses were in the right order of magnitude for a half-time of 5 to 6 hours. Accordingly, we decided to change to a 10 mg. per kilogram dose on a four-hour schedule. Electrocardiograms taken three and five hours after starting quinidine showed no change.

A serum level of 10.4 mg. per liter was obtained 12 hours after quinidine therapy was begun; electrocardiograms at this time showed regular rhythm. We elected at this point to continue quinidine with electrocardiographic tracings before each dose. At the end of 24 hours of quinidine administration the child showed no intolerance to the drug, the electrocardiograms continued to show regular rhythm with no evidence of undesirable quinidine effect, and the serum level was 11.5 mg. per liter.

During the next two days electrocardiographic tracings remained stable while the serum level gradually rose to a level of 14.8 mg. per liter (72 hours after the drug was started). Quinidine was then discontinued, and the normal sinus rhythm was present at the time of discharge two weeks later.

Unfortunately, events which occurred during and after therapy made it seem likely that the child was sensitive to quinidine. After 48 hours of quinidine administration there was evidence of bleeding from the lower gastrointestinal tract. Physical examination at this time revealed a few tiny bleeding points in the nasal mucosa; there were no purpura. A smear of peripheral blood was suggestive of decrease in platelet number, while the platelets seen appeared larger than normal.

Quinidine was continued for another 24 hours; the child was watched closely for any further evidence of bleeding. Platelet count at the conclusion of therapy (72 hours after beginning) was 85,000 (normal 300,000).

Because the cardiac response to quinidine had been so favorable and the possibility of future need for quinidine therapy was a real one, it was our opinion that further investigation of the possible hypersensivity to quinidine should be carried out. The patient's serum was found to have platelet agglutinins when her serum was combined with platelets and quinidine. She was also given a test dose of 10 mg. of quinidine sulfate. Before the test dose of quinidine the palatelet count was 285,000. Two and one-half hours after the dose was given the count dropped to 170,000 and 18 hours later the count was 185,000. The counts were done by phase microscopy.*

DISCUSSION

Since the method used in this study measures quinidine metabolites as well

*These data were kindly supplied by Dr. William Harrington of the Department of Medicine and with the help of Drs. Barbara Jones and W. G. Klingberg of the Department of Pediatrics.

as quinidine, the metabolism of the drug becomes a question of some importance. Unfortunately, the picture is by no means complete. Various observers have noted that only a small amount (about 15 per cent) of quinidine is excreted unchanged.[1, 16, 19] Brodie and co-workers[1] found that the principal metabolite of quinidine is a nonphenolic monohydroxy derivative. An enzyme system has been found in rabbit liver which is primarily concerned with the metabolism of cinchona alkaloids. But questions regarding the relative biologic activity of various quinidine metabolites remain unanswered. Since the possibility exists that the cardiac effects of the drug are produced by an intermediate metabolite, rather than by quinidine itself, there can be no particular disadvantage in a method of serum quinidine analysis which measures quinidine plus metabolites.

The problem of what constituted effective levels must await clinical trial in children with arrhythmias. Various observers have noted conversion to sinus rhythm at levels of 4 to 10 mg. per liter in adults with auricular fibrillation.[12, 16, 18] An 8-year-old child with persistent auricular flutter treated with quinidine at St. Louis Children's Hospital in 1953 showed conversion to sinus rhythm on two successive occasions when the quinidine serum level reached 16 mg. per liter as measured by the direct method.* In the case described in this paper conversion to sinus rhythm occurred in a 4-year-old child at a serum level of 10 mg. per liter.

The question of whether cardiac patients in congestive heart failure are able to metabolize and excrete quinidine at a "normal" rate is of considerable importance.[6] In a study of this problem in adults Swisher and associates[18] found that cardiac patients in congestive failure reached the same peak serum levels of quinidine as normal controls, but showed a slowed decline in levels after the drug was stopped. It should be noted that the extraction method (measurement of quinidine only) was used to determine levels in their study. It will be remembered we observed a slowly rising serum level during the seventy-two-hour period of quinidine therapy in a child with congenital heart disease, impure auricular flutter, and early cardiac failure. If we assume that the rising level was a reflection of quinidine metabolites measured, then the possibility of slowed metabolism or delayed excretion of metabolites in the patient with cardiac failure must be considered.

The relative insolubility of quinidine sulfate produced some problems peculiar to the age group to which it was administered. For example, it was found that the drug tended to precipitate out when preparations of quinidine sulfate or hydrochloride were diluted with saline for intramuscular use, and the 1 per cent solution necessary to effect solubility of the drug often meant that doses of 5 c.c. or more would have to be given. This difficulty was overcome by using quinidine gluconate, which may be diluted to any concentration. Since quinidine sulfate is readily soluble in alcohol (1 Gm. dissolves in 10 c.c. of alcohol), an oral preparation of the drug in an alcoholic vehicle was found to be useful, with ease of administration and accuracy of dosage being the chief ad-

*Data courtesy Dr. Del Harris.

vantages. The only possible disadvantage would be the need for fresh preparations because of evaporation.

In the course of this study two children with elevated temperatures received quinidine (one child had a temperature of 39° C., the other had 40° C.). In both cases subsequent levels were from two to three times higher than the expected value. It may be postulated that the increased metabolic rate which accompanies fever may result in such an increase in rate of metabolism of quinidine that excretion of metabolites lags behind. This appears to be borne out in one case in which simultaneous analysis by extraction and direct method was carried out: a blood sample collected when the child was afebrile showed 46.5 per cent metabolites, while a sample taken while he had fever contained 71 per cent metabolites. Since fever is a relatively frequent event in children, the relationship between fever and quinidine serum levels needs further investigation.

A second problem of interest relates to the fate of quinidine in patients with hydrocephalus. A total of eleven infants with hydrocephalus of varied etiology were given quinidine. Seven of these showed a delay (as compared with the majority) of one to two hours in reaching the maximum level, suggesting that the blood-brain barrier was permeable to quinidine. It would be of interest to investigate quinidine levels in the cerebrospinal fluid in children, since at least one observer has noted that little quinidine can be recovered from the cerebrospinal fluid of adults.[17]

A degree of individual variation in blood levels was noted, even within the age group tested. Similar variation has been observed in adults, but it has also been noted that uniform levels will be obtained if any individual is retested. In the present series ten children were retested; levels were approximately the same in successive determinations in eight instances.

In discussing half-time we suggested that half-time of quinidine approaches that seen in the adult as the child grows older. Certainly, the dosage requirement goes down as the child grows older. For example, in the infant group a dosage schedule of 7.5 mg. per kilogram every two hours gives a maximum serum level of about 9 mg. per liter, while an 8-year-old child being treated on the above dosage schedule maintained a serum level of 27 mg. per liter.

How should a child, perhaps 5 years old, be treated? To a certain extent such treatment would be on a trial-and-error basis, but with information gained from a minimum of blood levels a dosage schedule which would give predictable blood levels could be worked out fairly readily. We would suggest the following procedure for oral dosage: begin the patient on a 5 mg. per kilogram dose every two hours. Blood levels should be run one hour after the second and third doses, and should represent about half the maximum to be attained. At this point dosage could be adjusted up or down, as indicated. If the dosage used were in the correct range, a four-hour schedule could be started two hours after the third dose. The blood level should be checked at twelve hours, and again at twenty-four hours after starting therapy. By this time a plateau would have been reached, and there should

be no increase in blood levels, as long as dosage and interval were maintained.

We have had no experience with multiple doses of intramuscular quinidine, but do know that the half-time is three hours in infants and that a three-hour dosage schedule would probably be necessary to maintain uniform blood levels. We would begin with the same dose (5 mg. per kilogram) and run serum levels to help chart our course of treatment.

SUMMARY

1. The foregoing study was carried out to obtain information about quinidine dosage in children, utilizing serum levels of the drug.

2. A method for determining serum quinidine concentration which is simple, can be run on capillary blood samples, and is sufficiently accurate for clinical use has been presented.

3. On the basis of body weight, infants and young children appear to require relatively larger doses of quinidine than adults.

4. From information obtained in studies using single and multiple doses of quinidine, a tentative dosage schedule requiring a minimum of blood levels has been worked out.

5. A case has been presented in which the suggested dosage plan was successfully utilized to treat a 4-year-old child with congenital heart disease who had developed cardiac arrhythmia (impure auricular flutter).

6. Results which tended to vary from the normal were noted in patients with fever, with congestive heart failure, and with hydrocephalus; further work in these areas would be of particular interest.

We wish to express our gratitude to Drs. David Goldring and Oliver Lowry for their interest, encouragement, and valuable advice.

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International Virus Research and Immunization

(From the chapter on "Small Pox" in the English translation [1776] of Rosen von Rosenstein's *Diseases of Children* published in Sweden in 1771.)

However, the surest method to prevent the dangerous effect of the small-pox, is to inoculate it on children, while they are still young, and in the manner as is now customary in *England.*†

†The famous lady *Mary Wortley Montague* is the first who introduced the inoculation of small-pox into Europe. She had her son inoculated at *Constantinople,* at the age of six years; and her daughter inoculated in 1722, after their return to *England.* However, for the satisfaction of the court, the experiment was first made on six convicts, who were successfully inoculated, and thereby escaped their sentence of death. . . .

It is both strange and unaccountable to find how many means have been taken in *England,* by those who were against the inoculation, in order to deter the nation from it. . . .

The inoculation of the small-pox has met with no declared enemy in *Sweden,* but most likely has some secret opposer, . . . In the year 1761, several houses were mentioned, and which it was said that inoculated children had got a severe small-pox again. I went on purpose to visit both the parents and the children, and found, on enquiry, that all was an impudent fiction; this, and all the inventions employed in *England,* have persuaded me that the *cases* which are related from other countries against the inoculation, may be equally false and uncertain.

INCIDENCE OF PERTUSSIS IN VACCINATED AND UNVACCINATED CHILDREN

Report of 100 Cases Followed in a Child Health Clinic

Vita R. Jaffe, M.D.
Washington, D. C.

PERTUSSIS, once a serious disease of childhood, has in recent times become considered as uncommon and unimportant. Credit for this is given to the widespread use of immunization with pertussis vaccine. It has been assumed generally that immunization reduces the number of cases and the severity of the disease, although complete protection against pertussis by immunization has never been claimed. Statistics of reported communicable diseases disclose that whooping cough is uncommonly reported.

For this reason it was surprising to discover a large number of pertussis cases in a community with a good immunization program.

CASES FOUND

During the period from Feb. 1, 1953, to March 15, 1954, 100 culturally proved cases of pertussis were observed at two child health clinics of the Department of Public Health of the District of Columbia. These clinics are for preschool children, the overwhelming majority of whom are Negroes from a poor socioeconomic group. Only a small number of the sick children from the area are seen at these child health clinics as there are other facilities available at hospitals for sick children.

VACCINATION STATUS

Almost all of these children had been immunized at a district Department of Public Health child health clinic, and an accurate record of their immunization was available. The records of the children immunized elsewhere were obtained whenever possible. The immunization procedure during most of this period was to give at monthly intervals four injections of 0.5 c.c. of diphtheria and tetanus toxoid and pertussis vaccine combined, alum-precipitated, starting at 3 months of age. If the immunizations were not started until 6 months of age, or later, three injections at monthly intervals were given, two injections of 0.5 c.c. of the preparation and 1.00 c.c. for the third injection. About one year later a booster dose of 1.0 c.c. was given, and then a second booster at about 4 years of age.

CLINICAL DIAGNOSIS

History.—In the series of cases under consideration a presumptive diagnosis of pertussis was made almost entirely on the basis of the history given by the mother. Pertussis was suspected when the history disclosed a bad night cough of over one week's duration which was getting worse

From the Department of Public Health of the District of Columbia, Bureau of Maternal and Child Health.

while the child remained afebrile. Additional symptoms frequently described were spells of coughing and strangling, and in some cases gagging, vomiting, and whooping.

Physical Examination.—The physical examination was helpful, chiefly in a negative sense, that is, the diagnosis of pertussis was considered more likely if there was nothing found on physical examination to account for the above symptoms. Only in the more severe cases was any coughing observed in clinic, since these children did most of their coughing at night.

WHITE BLOOD COUNT

White blood counts were made on a number of the children. These counts were not characteristic of pertussis as given in textbooks. The total white blood cell count ranged from 8,950 to 14,200, with a lymphocyte count of 36 to 80 per cent. In other words, the white blood count was of no help in making the diagnosis, and was what one would expect to find in an upper respiratory infection.

BACTERIOLOGICAL PROOF

Every one of these 100 cases had a positive cough plate culture. Only one plate was used per child.

All of the cough plate cultures were taken and examined by Dr. J. B. Holland, Chief of the Division of Bacteriology of the District of Columbia Department of Public Health. Dr. Holland's report of his method and findings states:

"These cultures were prepared by holding a plate of medium (15 c.c.) directly in front of and about 6 inches from the child's mouth during paroxysm. Paroxysms are produced by exerting some pressure with the thumb, on the trachea, just below the cricoid cartilage.

"The medium used is Bordet-Gengou, potato, blood agar (modified), containing 18 per cent defibrinated blood. These plates after exposure are incubated at 37° C. for seventy-two hours.

"The characteristic colonies (small dome-shaped, moist and pearly white, surrounded by a slight darkened zone) are fished off and subcultured on plain agar plates and also on Bordet-Gengou agar. Colonies failing to grow on the plain agar plates are fished from the Bordet-Gengou subcultures, stained by Gram's method, and if gram-negative, morphologically resembling *Hemophilus pertussis*, they are then agglutinated against anti-*hemophilus pertussis* serum, when available."

Dr. Margaret Pittman of the Laboratory of Biologics Control, of the National Institutes of Health, confirmed the bacterial identification of *H. pertussis* from six consecutive cases.

Dr. Holland reports that from Jan. 1, 1953, through April, 1954, a total of 379 such cultures were processed, of which 144, or slightly over 38 per cent, were found positive. This represents the total number of cultures submitted by a number of district health department clinics as well as by a number of private physicians.

TYPE OF CASES

Most of the cases in this series of 100 under consideration were mild. The only significant complication was otitis media in one child. None of the children were hospitalized. All recovered.

TREATMENT

Treatment consisted only of cough syrup and other symptomatic measures in most cases. All infants under

one year received one or two injections of Hypertussis* with good results. In those cases where additional treatment was necessary, Aureomycin (10 mg. per pound per twenty-four hours) was found effective. Hypertussis was given prophylactically to all exposed children under one year of age.

children had pertussis confirmed by positive cough plate in spite of completed schedules of vaccination.

RELATION OF VACCINATION TO SEVERITY OF DISEASE

The clinical impression was that vaccination did not affect the severity of

TABLE I. AGE AND VACCINATION STATUS OF CHILDREN WITH POSITIVE COUGH PLATES FOR PERTUSSIS

AGE	TOTAL	VACCINATION STATUS			
		NONE	SOME	3 OR 4 DPT INJECTIONS	3 OR 4 DPT INJECTIONS PLUS 1 OR 2 BOOSTERS
Under 3 mo.	4	4			
3-5 mo.	9	6	3		
6-11 mo.	20	3	8	9	
12-17 mo.	10	1	1	8	
18-23 mo.	12	1	2	9	1
2 yr.	15	1	1	13	4
3 yr.	13	2	1	10	5
Over 4 yr.	17	2	–	15	12
Total	100	20	16	64	22

TABLE II. THE OCCURRENCE OF PERTUSSIS AND VACCINE STATUS OF CHILDREN UNDER 10 YEARS OF AGE HAVING HISTORY OF NO PRIOR PERTUSSIS AND NO PROPHYLACTIC HYPERIMMUNE SERUM WHO WERE ASSOCIATED IN A FAMILY HOUSEHOLD WITH A CASE OF PERTUSSIS IN A SIBLING*

AGE (YEARS)	OLDEST CASE EXCLUDED	VACCINE STATUS OF SIBLINGS IN HOUSEHOLD							
		VACCINATED 3 OR MORE DOSES		VACCINATED LESS THAN 3 DOSES		NO VACCINATIONS		TOTAL	
		CASES PER PERSONS	PER CENT	CASES PER PERSONS	PER CENT	CASES PER PERSONS	PER CENT	CASES PER PERSONS	PER CENT
Under 1	4	5/5	100	6/7	87	8/9	89	19/21	90
1	2	9/11	82	3/3	100	2/2	100	14/16	88
2	8	12/14	86	2/2	100	0/0	–	14/16	88
3	11	4/8	50	0/1	–	3/4	75	7/13	54
4-9	25	7/24	29	0/2	–	2/5	40	9/31	29
Total	50	37/62	60	11/15	73	15/20	75	63/97	63

*At least one bacteriologically confirmed case in each household.

VACCINATION STATUS

In Table I is given an analysis of the 100 cases according to age and pertussis vaccination. It will be seen that 64 per cent of these children had three or four injections of diphtheria and tetanus toxoids and pertussis vaccine, alum-precipitated, and 22 per cent of the patients also had one or two booster injections. Thus, sixty-four

the disease. Age seemed a more important factor. The disease was more severe in younger children.

ANALYSIS OF HOUSEHOLDS

To determine the incidence of pertussis in a specific group of children an analysis was made of all the households involved in which there were more than one child under 10 years of age who had no prior history of pertussis and no prophylactic hyperimmune

*Antipertussis human serum made by Cutter Laboratories.

serum and who were exposed to a case of pertussis in a sibling. In each household there was at least one bacteriologically confirmed case. The results are shown in Table II.

VALUE OF VACCINATION

Table II shows that although there were fewer cases of pertussis in vaccinated children, the difference is not statistically significant. In the sixty-two vaccinated siblings, thirty-seven, or sixty per cent, developed pertussis. In the fifteen children who had partial vaccination, eleven, or 73 per cent, developed pertussis. Of the twenty unvaccinated siblings, fifteen, or 75 per cent, developed pertussis. It is of interest to compare this observation with those of others on the secondary attack rate in vaccinated children as compared with unvaccinated children in the same household. Kendrick[1] found rates of 36.4 and 92 per cent respectively, and Bell[2] 30.6 per cent (of 109 children) and 87 per cent (of 115 children), respectively.

POTENCY OF VACCINE

One reason for the poor protection offered by vaccination in this series of cases may be due to the variable potency of the vaccine used. It was estimated that the children would have been vaccinated with lots of vaccine purchased by the District of Columbia Health Department between April 17, 1950, and Nov. 13, 1953. During this time the lots of triple antigen were obtained from two licensed manufacturers and the lots contained 20,000 or 30,000 million bacteria per cubic centimeter, and 2.0 and 1.5 c.c., respectively, was recommended for the total immunizing dose. This period was before the recent change which requires that each lot of pertussis vaccine contain 12 units per total immunizing dose Pittman.[3] However, through the cooperation of the two manufacturers, Dr. Margaret Pittman of the Laboratory of Biologics Control, National Institutes of Health, was able to obtain an estimate of the potency of 90 per cent of the 12,583 containers of vaccine purchased during this period. Of these, only 41 per cent had 12 units per total dose, 34.5 per cent had values as low as 5.5 to 6.5 units, while the remainder ranged from 7.3 to 9.6 units. It is hoped that in the future, under the new potency requirements for pertussis vaccine, better protection will be provided by vaccination than was observed in this series of cases.

SUMMARY

It would appear from this study that pertussis is not an uncommon disease and that even in vaccinated children it continues to occur. It may be that the diagnosis is not being made either because of its atypical mild form at the present time or because it is cured by one of the broad-spectrum antibiotics before its full symptomatology erupts.

I am grateful to Drs. Joseph Bell and Margaret Pittman, both of the National Institutes of Health, for their helpful suggestions, and to the people in the District of Columbia Department of Public Health for their assistance.

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RETROGRADE AORTOGRAPHY IN THE DIAGNOSIS OF CONGENITAL HEART DISEASE IN INFANTS

EDWARD B. SINGLETON, M.D., DAN G. MCNAMARA, M.D., AND DENTON A. COOLEY, M.D.
HOUSTON, TEXAS

IN MANY instances diagnosis of congenital lesions of the heart and great vessels in infants may be established by ordinary clinical methods, including routine fluoroscopy and radiography. Occasionally, however, more specialized radiographic procedures must be used. This is especially true in the diagnosis of certain cardiac lesions often encountered in the infant age group. At this age great reliance cannot be placed on physical findings and the abnormality has not been present for a sufficient length of time to produce the characteristic radiographic configuration of the heart seen in older patients. In addition, the relatively large thymus of the infant often obscures the cardiac boundaries, making radiologic interpretation more difficult. Although cardiac catheterization and angiocardiography are extremely useful in diagnosis of most congenital abnormalities of the heart, they may be inadequate in demonstrating the exact nature of extracardiac shunts and in detecting abnormalities of the aortic arch. Keith and Forsyth[1] have described a technique for visualization of the aortic arch in infants by the retrograde injection of radiopaque medium into the left brachial or radial artery. By this method abnormalities of the thoracic aorta or communications between the aorta and pulmonary artery can be demonstrated. We have found this procedure useful in establishing or excluding the diagnosis of patent ductus arteriosus, aortic septal defect, coaretation of the aorta, and true truncus arteriosus. Since many of these abnormalities are surgically correctable, accurate identification of the lesion is of considerable importance.

The differential diagnosis in infancy between a high ventricular septal defect and a large patent ductus arteriosus in which the continuous murmur has not developed is frequently difficult because of features common to both conditions. If cardiac enlargement or other signs of congestive failure are present, diagnosis becomes urgent in order that corrective surgery may be performed. Cardiac catheterization is at times useful in differentiating these two conditions, but the procedure in infants is technically difficult and furthermore the proximity of the location of the shunt in the two lesions may lead to errors in interpretation. Retrograde aortography, however, will demonstrate that there is communication between the aorta and pulmonary circulation in patent ductus arteriosus, while in ventricular septal defects no such communication is

From the departments of radiology, pediatrics, and surgery of Texas Children's Hospital and Baylor University College of Medicine.

Aided in part by a grant from the Houston District Chapter of the Texas Heart Association.

shown. Occasionally the aortic-pulmonary shunt is produced by an aortic septal defect rather than by patent ductus arteriosus and the aortographic findings may be indistinguishable. However, the surgical approach in both lesions is similar and accurate radiographic differentiation is probably unnecessary.

In true truncus arteriosus large pulmonary arteries arise from the aorta, usually from the ascending portion. Consequently, adequate filling of the ascending aorta by retrograde aortography will result in visualization of the contrast material in the pulmonary circulation. Without knowledge of the clinical findings, the aortogram may resemble that of patent ductus arteriosus or aortic septal defect, but final differentiation of truncus from these other lesions may be obtained by angiocardiography. This demonstrates early visualization of the large truncus and at the same time fails to demonstrate a pulmonary artery arising from the right ventricle.

Although venous angiocardiography may demonstrate coarctation of the aorta, the contrast material is often too dilute by the time it reaches the aorta to accurately delineate the character of the constriction. This is particularly true if there is an associated intracardiac left-to-right shunt. Retrograde aortography, on the other hand, will demonstrate the site of coarctation more accurately,[2] and if patent ductus arteriosus is also present, opacification of the pulmonary arteries will be evident. In that type of coarctation reported by McGregor and Medalie,[3] in which none of the peripheral pulses are palpable, differentiation from aortic stenosis may be impossible without retrograde aortography. Furthermore, whenever operation is indicated for coarctation of the aorta in infants, intractable congestive failure is usually present and venous angiocardiography is more hazardous than aortography.

TECHNIQUE

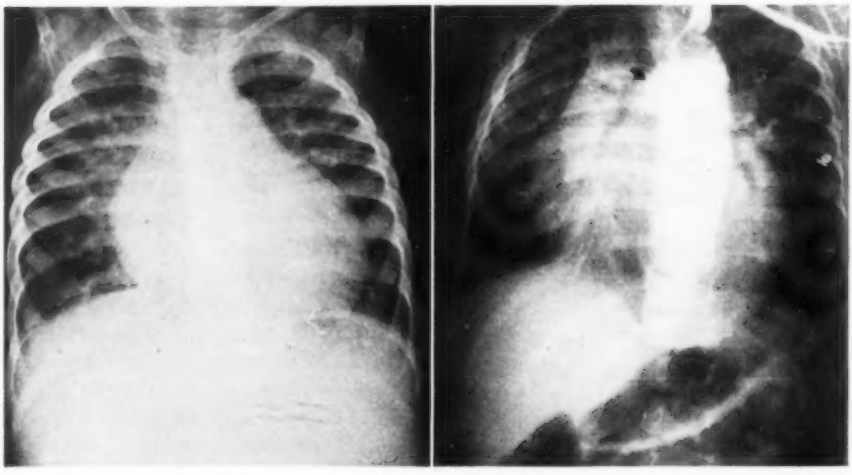
Retrograde aortography may be performed in infants under local anesthesia. Oral intake should be withheld four hours before the procedure, and mild sedation is advisable, consisting of chloral hydrate or pentobarbital suppository administered thirty minutes previously. With the patient lying supine on the fluoroscopy table, the inner aspect of the left upper arm is prepared and draped with sterile towels. After locating the brachial arterial pulsation by palpation, the skin and subcutaneous tissues are infiltrated with procaine hydrochloride, 1 per cent solution. The artery is exposed through a 2 cm. longitudinal incision, carefully moving the median nerve and veins aside. Ligatures are passed around the artery above and below the point of insertion of the needle. A small opening is made in the artery and a 19 gauge Lindemann's needle with stylet is inserted. The patient is then placed over the cassette changing device, usually in the right posterior oblique position. Urokon, 30 per cent solution, is used for the injection, the dosage of which is determined on the basis of 1 c.c. per kilogram of body weight. With the cassette changer in motion at a speed of 2 frames per second, the contrast material is rapidly injected. At the completion of the injection the arteriotomy incision is closed with a 6-0 arterial silk suture and bleeding

is minimal. The skin is sutured with interrupted silk sutures and a sterile dressing is applied.

ILLUSTRATIVE CASES

Patent Ductus Arteriosus

CASE 1.—L. C., an 11-month-old white female, was admitted to the Texas Children's Hospital on March 9, 1955. The patient was apparently normal until the age of 6 months, when the onset of dyspnea and "mottling" of the skin was noted. At 10 months of age she had pneumonia and congestive heart failure requiring digitalization. On examination, the patient was poorly nourished and weak. The heart was enlarged, and there was a palpable thrill over the sternum. Harsh systolic murmur was audible to the left of the sternum in the third intercostal space and was questionably continuous in character. A diastolic gallop was present at the apex and rhonchi were heard over the lung fields. Blood pressure was 120/30 mm. Hg. The liver was moderately enlarged. The electrocardiogram showed a first-degree A-V block and left ventricular hypertrophy. Fluoroscopy and radiography of the chest showed considerable enlargement of the heart, predominantly left ventricular. The undivided pulmonary artery segment was prominent and pulmonary vascular markings were increased (Fig. 1, *A*). Retrograde aortography showed visualization of the main pulmonary artery, simultaneously with the arch of the aorta (Fig. 1, *B*). At thoracotomy on March 14, a patent ductus, 12 mm. in diameter, was divided and sutured. Recovery was uneventful and the patient has remained well.



A. *B.*

Fig. 1.—Roentgenograms of chest in Case 1. In the posteroanterior projection, *A*, is demonstrated cardiac enlargement and increased vascularity. Retrograde aortogram, *B*, shows opacification of the pulmonary artery segment by contrast medium, establishing the diagnosis of patent ductus arteriosus.

Comment.—Although a continuous murmur was heard on one occasion, it was not sufficiently typical to warrant exploration without a definite diagnosis being established. Severe congestive failure made immediate surgery mandatory in this infant.

Coarctation of the Aorta

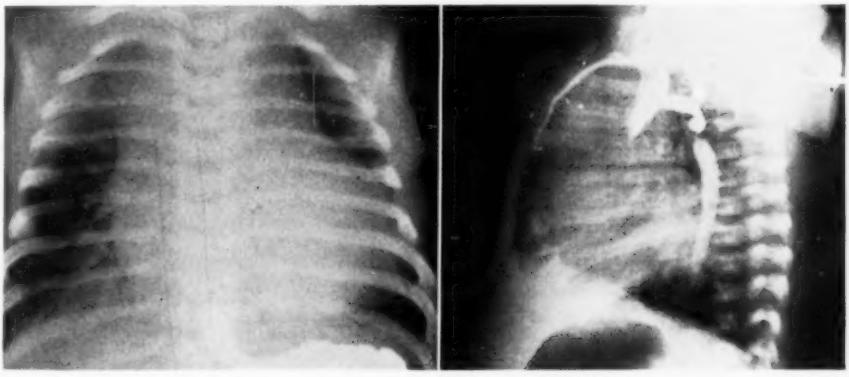
CASE 2.—C. P. M., a 4-week-old white infant, was admitted to Texas Children's Hospital on Jan. 8, 1955,

with dyspnea and cyanosis which developed forty-eight hours prior to admission. Examination revealed an acutely ill infant in severe respiratory distress, grade 2 cyanosis of the nails, and generalized "mottling" of the skin. The heart was enlarged and a faint systolic murmur was audible along the left sternal border with a diastolic gallop at the apex. The liver edge was palpable 3 cm. below the costal margin. Pulsations in the arms were easily palpable but were absent in the lower extremities. By the "flush" technique the blood pressure in the arms was 136 mm. Hg, and in the legs 72 mm. Hg. Electrocardiograms showed left ventricular hypertrophy. Fluoroscopy and radiography of the chest showed marked cardiac enlargement, predominantly left ventricular (Fig. 2, *A*). There was also definite evidence of left atrial enlargement. Pulmonary vascularity was prominent but had the appearance more suggestive of passive venous congestion than increased blood flow. Retrograde aortograms showed a typical coaretation of the aorta located just distal to the subclavian artery (Fig. 2, *B*). The opacified internal mammary artery was enlarged. At operation on January 11, the constricted segment of aorta was resected and end-to-end anastomosis performed. Recovery was uneventful. The patient was discharged from the hospital ten days later and has remained well.

Comment.—Even though the clinical diagnosis of coaretation of the aorta was apparent in this case, the demonstration of the exact type of coaretation confirmed the diagnosis and facilitated the surgical procedure.

True Truncus Arteriosus

CASE 3.—D. P., a 2-year-old male infant, was admitted to Texas Children's Hospital on Dec. 12, 1954, with progressive dyspnea. During the first few months of life he was noted to have a forceful heartbeat and difficulty in breathing. At 9 months of age he developed pneumonia, and a cardiac murmur was discovered. On examination the infant was underdeveloped, the heart was enlarged, and forceful pulsations were noted. There was a grade 3 systolic murmur along the left sternal border and the pulmonary second sound was accentuated and split. A mid-diastolic gallop was audible at the apex. Blood pressure in the upper extremities was 100/75 mm. Hg and in the lower extremities 100/80 mm. Hg. The electrocardiogram revealed

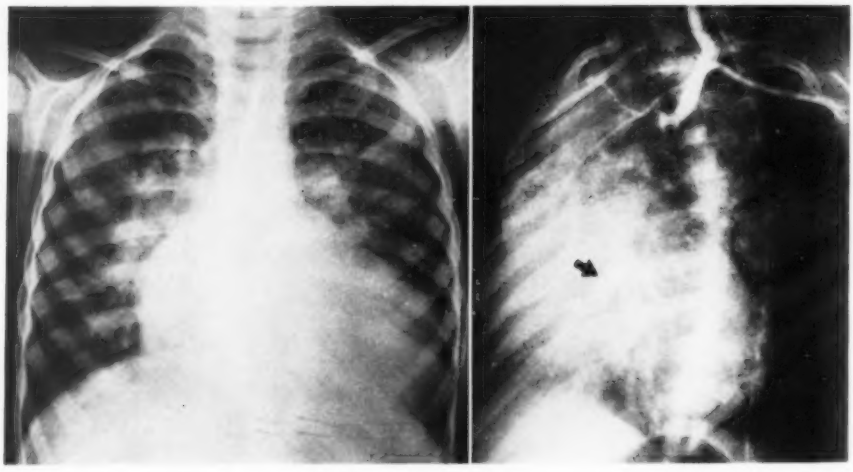


A. *B.*

Fig. 2.—Roentgenograms of chest in Case 2. Posteroanterior view, *A*, demonstrates the marked cardiac enlargement and passive venous congestion. Retrograde aortogram, *B*, shows the area of coarctation distal to the origin of the left subclavian artery.

right ventricular hypertrophy and first degree A-V block. Fluoroscopy and radiography of the chest demonstrated cardiac enlargement, particularly of the right atrium and right ventricle (Fig. 3, *A*). The pulmonary artery segment was concave. Marked increase in pulmonary vascularity was noted and minimal hilar pulsations were seen fluoroscopically. Barium swallow showed evidence of slight enlargement of the left atrium. The superior mediastinum was narrow in both anteroposterior and oblique views. Cardiac catheterization showed an increase in oxygen content in the pulmonary artery and the catheter was thought to enter the aorta through the pulmonary trunk. Femoral arterial saturation was 84 per cent. Aortography was performed, and although the aortic arch was not well seen by this procedure, there was opacification of the pulmonary vessels (Fig. 3, *B*). In view of the clinical and other radiographic findings, this was interpreted as representing truncus communis rather than patent ductus arteriosus. Angiocardiograms confirmed this impression by demonstrating early filling of the aorta with evidence of pulmonary vessels arising from the ascending portions of the aorta.

Comment.—Although the evidence of right ventricular hypertrophy and arterial unsaturation suggested truncus arteriosus, it was impossible from the catheterization data to definitely exclude patent ductus arteriosus or septal defect. Retrograde aortography showed communication between the aorta and pulmonary circulation and angiocardiography established the diagnosis of truncus arteriosus.



A. *B.*

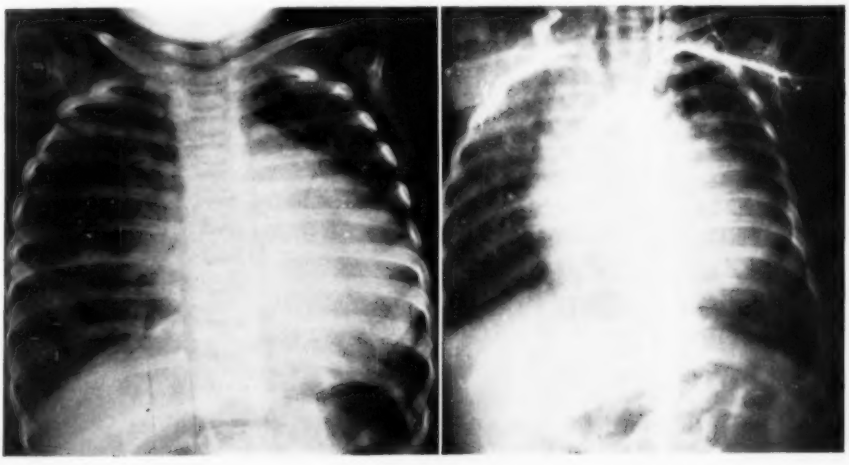
Fig. 3.—Roentgenograms of the chest in Case 3. Posteroanterior chest film, *A*, shows a narrow cardiac waist, cardiac enlargement, and increased pulmonary vascularity. Retrograde aortogram, *B*, shows opacification of pulmonary arteries (arrow).

Ventricular Septal Defect

CASE 4.—D. M., a 4-month-old white female infant, was admitted to Texas Children's Hospital on Feb. 23, 1955, because of failure to gain weight. Physical examination revealed a frail infant. A loud systolic murmur was audible over the left third and fourth intercostal spaces accompanied by an accentuated pulmonary second sound. An electrocardiogram showed combined ventricular hypertrophy and a first-degree A-V block. Fluoroscopy and

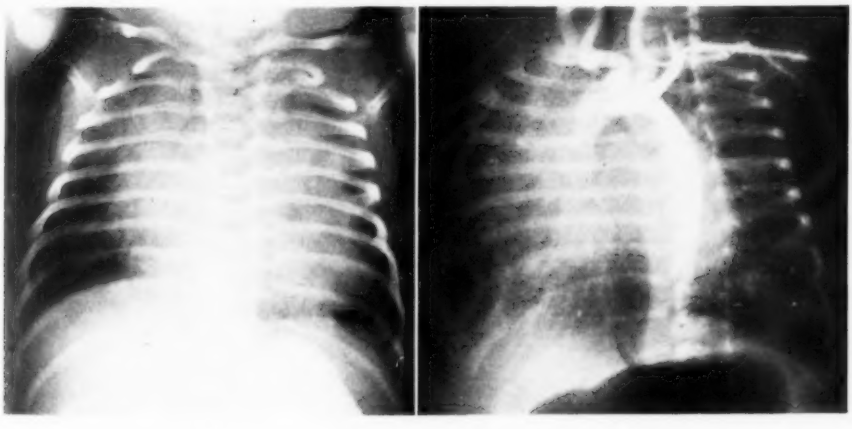
radiography of the chest showed moderate cardiac enlargement with dilatation of the left ventricle and left atrium (Fig. 4, *A*). Retrograde aortography showed a normal aortic arch without evidence of communication with the pulmonary circulation (Fig. 4, *B*).

Comment.—It was impossible to determine from the clinical data and electrocardiograms whether this patient had an interventricular septal defect or patent ductus arteriosus. Retrograde aortograms were of value in ruling out the latter condition.



A. *B.*

Fig. 4.—Roentgenograms of chest in Case 4. Posteroanterior chest film, *A*, shows marked enlargement of the left ventricle and increased pulmonary vascularity. Retrograde aortogram *B*, demonstrates a normal aorta without opacification of the pulmonary circulation, thereby excluding patent ductus arteriosus.



A. *B.*

Fig. 5.—Roentgenograms of chest in Case 5. Posteroanterior chest film, *A*, shows cardiac enlargement. Retrograde aortogram, *B*, shows a normal aorta, thereby excluding coartation of the aorta.

Aortic Stenosis

CASE 5.—R. V. S., a 25-day-old white male infant, was admitted to Texas Children's Hospital on April 11, 1955, because of respiratory distress and perioral cyanosis of six days' duration. Physical examination revealed an infant in marked respiratory distress who was cyanotic while crying. No murmur or thrill was detected, but a diastolic gallop was audible at the apex. The eyelids were edematous, and inspiratory râles were heard over each lung. The peripheral pulses were not palpable except for a questionable faint pulse in the right arm. Electrocardiogram showed slight left ventricular hypertrophy. Fluoroscopy and radiography disclosed marked cardiac enlargement, predominantly left atrial and left ventricular (Fig. 5, *A*). Pulmonary vascular markings were prominent and had the appearance of venous congestion. Minimal right pleural effusion was present. Retrograde aortography showed a normal aorta without communication with the pulmonary circulation (Fig. 5, *B*). The patient died in congestive failure on April 21, 1955. Autopsy revealed an aortic valvular stenosis with a valvular orifice approximately 2 mm. in diameter.

Comment.—Because of absence of pulsations in the peripheral arteries, a diagnosis of aortic stenosis was considered, but an unusual type of coarctation of the aorta could not be excluded without aortography.

SUMMARY

The diagnosis of congenital cardiac malformations in infants is frequently difficult. Accurate and prompt diagnosis is often essential in order to determine if the abnormality is surgically correctable. Retrograde aortography is a useful procedure in establishing or excluding the diagnosis of several types of malformation, particularly patent ductus arteriosus, aortic septal defect, coarctation of the aorta, and truncus communis from other lesions such as interventricular septal defect and congenital aortic stenosis, where the clinical findings may be similar. Five cases are presented, illustrating the usefulness of this method.

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STUDIES OF 17-HYDROXYCORTICOSTEROIDS

XIII. Effects of Salicylate Intoxication in Rheumatic and Nonrheumatic Subjects

Alan K. Done, M.D., Robert S. Ely, M.D., Richard B. Raile, M.D., and Vincent C. Kelley, M.D., Ph.D.
Salt Lake City, Utah

AN EARLIER publication[1] reported the consistent finding of markedly elevated circulating concentrations of 17-hydroxycorticosteroids (17-OHCS) in patients with salicylate intoxication and in guinea pigs given large doses of sodium salicylate. The present report presents additional observations concerning 17-OHCS plasma levels in nonrheumatic individuals following the accidental or suicidal ingestion of large amounts of salicylate and, in addition, introduces data from similar studies in patients with active rheumatic fever and superimposed iatrogenic salicylate intoxication.

SUBJECTS AND METHODS OF STUDY

The subjects included in this study are divided into three categories: (1) Four children and nine adults with clinically and chemically established salicylate intoxication, accidental or suicidal in origin. Data for seven of these individuals have been reported previously.[1] Each patient of this group was physically well prior to the ingestion of a toxic dose of aspirin, with the exception of one child (R. W., Table I) who had a mild upper respiratory infection; (2) eight children with active rheumatic fever and superimposed iatrogenic salicylate intoxication; and (3) five children and one adult who, following the ingestion of an unknown quantity of aspirin, were seen in the emergency ward and subjected to gastric lavage, but did not develop high plasma salicylate levels or symptoms of intoxication.

Plasma 17-OHCS concentrations were determined by the method of Nelson and Samuels[2] as modified by Eik-Nes, Nelson, and Samuels.[3] Plasma salicylate determinations during the early phase of the study were by the method of Brodie and associates[4]; more recent estimations were by the technic of Trinder.[5] Duplicate determinations have shown good agreement between values obtained by these two methods.

OBSERVATIONS

In Table I are shown data concerning plasma concentrations of 17-OHCS in thirteen nonrheumatic individuals with salicylate intoxication. Case histories of the seven patients whose initials are marked with an asterisk are reported in detail elsewhere.[1] The mean plasma concentration of 17-OHCS in normal children is nearly identical with that in normal adults, being 12.0 μg per 100 ml. (S.E.M.,

From the Departments of Pediatrics, University of Utah College of Medicine, Salt Lake City, and University of Minnesota, Minneapolis.

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1.29; S.D. 8.2) in the former[6] and 13.0 μg per 100 ml. (S.E.M., 0.4; S.D., 6.0) in the latter.[7] It can be seen that each of the nonrheumatic patients with salicylate intoxication had a markedly elevated circulating concentration of 17-OHCS. In general, the greatest elevations of steroid levels were found in the young children.

rushed to the emergency ward following the accidental or suicidal ingestion of unknown amounts of aspirin but had failed to develop clinical or laboratory evidences of intoxication by the time of sampling. Each of these individuals was subjected to gastric lavage, and blood samples were obtained for study immediately upon dis-

TABLE I. PLASMA 17-OHCS CONCENTRATIONS IN NONRHEUMATIC PATIENTS WITH SALICYLATE INTOXICATION

PATIENT	AGE AND SEX	DURATION, SALICYLATE ADMINISTRATION	DURATION, SALICYLATE INTOXICATION	SEVERITY SYMPTOMS OF SALICYLATE INTOXICATION	PLASMA SALICYLATE (MG./100 ML.)	PLASMA 17-OHCS (μG/100 ML.)
R. W.*	9 mo. M	5 da.‡	1 da.	+ + +	33	44.0
W. D.†	24 mo. M	4 hr.	2 hr.	±	66	58.2
K. S.*	29 mo. F	24 hr.	12 hr.	+ + +	32	48.2
A. G.*	29 mo. M	48 hr.	45 hr.	+ + + +	22	41.0
C. C.	15 yr. F	7 hr.	6 hr.	+ +	65	33.4
		27 hr.	26 hr.	+	46	27.1
D. G.*	16 yr. M	18 hr.	14 hr.	+ +	64	31.4
		38 hr.	34 hr.	+	19	7.3
A. N.†	17 yr. F	4 hr.‡	1 hr.	+ +	44	23.1
S. B.*†	17 yr. F	4 hr.	2 hr.	+	60	30.5
D. C.	19 yr. F	8 hr.	2 hr.	+	47	27.0
D. B.†	21 yr. F	4 hr.	1 hr.	+ + to + + +	88	49.0
J. G.*†	21 yr. F	2½ hr.	½ hr.	+ +	35	43.7
G. A.	25 yr. F	5½ hr.	½ hr.	+ +	64	20.6
		9 hr.	4 hr.	+	56	24.0
C. A.*	61 yr. F	18 hr.‡	8 hr.	+ +	51	33.4
		42 hr.	32 hr.	±	28	20.5

*Previously reported.[1]

†Gastric lavage immediately prior to study.

‡Continuous administration throughout the period indicated. In all other patients, salicylate was taken in single dose.

TABLE II. PLASMA 17-OHCS FOLLOWING ACUTE SALICYLATE INGESTION AND GASTRIC LAVAGE IN ABSENCE OF INTOXICATION AT TIME OF SAMPLING

PATIENT	AGE (YR.) AND SEX		INTERVAL SINCE INGESTION (HR.)	PLASMA SALICYLATE (MG./100 ML.)	PLASMA 17-OHCS (μG/100 ML.)
B. B.	2	M	2½	22	22.9
D. H.	3	M	2	17	6.2
B. S.	3	F	1	10	25.3
S. C.	4	M	1½	23	22.3
N. R.	4	F	2	20	16.1
K. E.	17	F	1	15	19.0

In an attempt to delineate the possible contribution to these increases in circulating 17-OHCS of the combination of such factors as excitement, gastric lavage, venepuncture, etc., determinations of 17-OHCS levels were made in six individuals who were

continuation of lavage. The data are shown in Table II. Moderate elevations of plasma 17-OHCS concentrations were found in some of these individuals, particularly the younger ones, but the levels found were not of the magnitude of those in patient

with salicylate intoxication. Moreover, review of Table I reveals no apparent relationship of gastric lavage to magnitude of 17-OHCS concentration in patients with salicylate intoxication. It should be noted (Table I) that only one of the children (patient W. D.) with salicylate intoxication was subjected to gastric lavage prior to sampling.

than the mean for normal controls but considerably lower than the levels seen in most of the nonrheumatic subjects with salicylate intoxication. Table IV presents the data for these patients in relation to duration of rheumatic symptomatology and, for purposes of comparison, lists mean plasma 17-OHCS concentrations for untreated rheumatic fever patients during corre-

TABLE III. PLASMA 17-OHCS IN PATIENTS WITH ACTIVE RHEUMATIC FEVER AND SALICYLATE INTOXICATION*

PATIENT	AGE (YR.) AND SEX	DURATION (DA.) RHEUMATIC FEVER	SEVERITY SYMPTOMS OF SALICYLATE INTOXICATION	PLASMA SALICYLATE (MG./100 ML.)	SALICYLATE THERAPY	PLASMA 17-OHCS (μG/100 ML.)
G. T.	13 M	6	++		4 Gm./da. for 4 da.	4.9
J. Mo.	7 M	7	++++	88	5⅓ Gm./da. for 2 da.	23.4
D. R.	11 M	13	++	37	8 Gm. over 36 hr. period	18.0
J. Me.	14 M	14	++		8 Gm. over 8 hr. period	7.9
C. J.	13 F	16	++	60	8 Gm./da. for 5 da.	1.9
L. M.	11 F	16	++	56	8 Gm./da. for 7 da.	6.0
T. F.	13 M	21	++	56	8 Gm./da. for 5 da.	2.2
W. S.	6 M	22	++	44	2⅔ Gm./da. for 6 da. 3⅓ Gm./da. for 4 da.	0.0

*Blood specimens were obtained within approximately eight hours after development of evidences of intoxication.

TABLE IV. PLASMA 17-OHCS (μG PER 100 ML.) IN RHEUMATIC PATIENTS WITH SALICYLATE INTOXICATION, IN RELATION TO PHASE OF RHEUMATIC FEVER (R. F.)

EARLY ACUTE* R. F.	TRANSITIONAL-PHASE† R. F.	WELL-ESTABLISHED ACTIVE‡ R. F.
4.9	18.0	1.9
23.4	7.9	6.0
		2.2
		0.0
Mean Plasma 17-OHCS in Untreated Rheumatics§		
23.1	11.8	5.9

*First week of rheumatic symptomatology.
†Second week of rheumatic symptomatology.
‡Active R. F. beyond second week.
§Data from reference 6.

Table III presents data concerning 17-OHCS concentrations in the plasma of patients with active rheumatic fever and salicylate intoxication. Six of the eight patients in this group were found to have circulating 17-OHCS concentrations which were lower than the mean for normal individuals. The two remaining subjects (J. Mo. and D. R.) had 17-OHCS levels which were higher

sponding phases of the disease. Review of these data indicates that *none* of the salicylate-intoxicated rheumatic subjects had plasma 17-OHCS levels which were appreciably higher than those seen in untreated rheumatics.

Fig. 1 presents a graphical comparison of 17-OHCS concentrations in subjects of each of the study groups. Individual points represent plasma 17-

OHCS concentrations in the subjects with salicylate intoxication or in the "lavage controls." The dotted line across each bar graph represents: in the columns for nonrheumatic subjects, the mean plasma 17-OHCS concentration in untreated control subjects; in the columns for rheumatic patients, the mean plasma 17-OHCS concentration in patients with untreated rheumatic fever of similar duration.

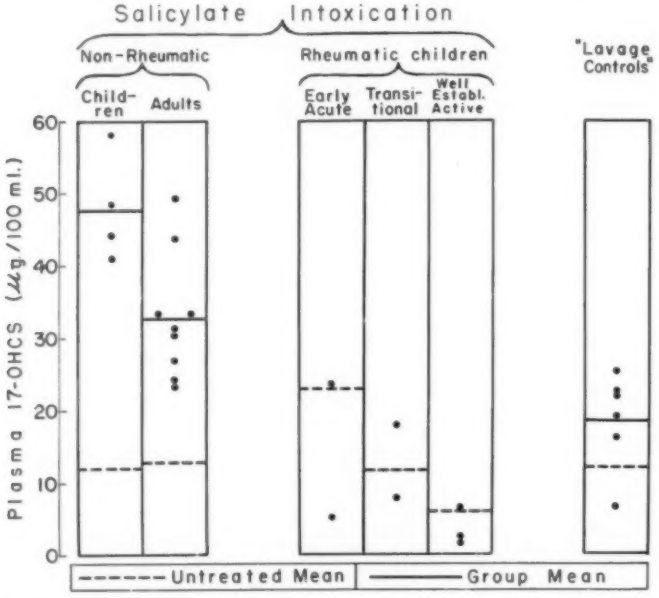

Fig. 1.—Plasma 17-OHCS in nonrheumatic and rheumatic patients with salicylate intoxication and in "lavage controls."

DISCUSSION

Data of the present report augment previously reported observations[1] of markedly elevated circulating 17-OHCS concentrations in nonrheumatic individuals with salicylate intoxication. To date, this has been a consistent finding. Possible explanations for these elevations have been discussed previously[1]; however, certain data of the present report offer additional information regarding salicylate-induced elevations of plasma 17-OHCS concentrations in previously normal individuals. Patients with subtoxic salicylate levels, who were subjected to gastric lavage and the other physical and emotional traumas experienced by the definitely intoxicated subjects, had plasma 17-OHCS concentrations which were elevated as compared with normal individuals, but much less elevated than patients of similar age with salicylate intoxication. It would appear that these factors have a role as nonspecific "stress" stimuli in producing increased steroid levels, but fall far short of accounting exclusively for the markedly elevated 17-OHCS concentrations seen in salicylate intoxication. This does not imply, however, that the effect of sali-

cylate intoxication, per se, in producing steroid elevations may not be one of nonspecific "stress."

In view of the uniformity of findings regarding 17-OHCS in patients with salicylate intoxication, the failure to find high 17-OHCS concentrations in similarly intoxicated subjects with active rheumatic fever was rather surprising. A definitive explanation is not available from the data at hand, but certain possibilities deserve discussion. These data might be interpreted as supporting the previously reported concept of relative adrenal-cortical insufficiency in patients with rheumatic fever.[8] That is, the stimulus to the pituitary-adrenal system occasioned by salicylate intoxication—whether a direct or a "stress" effect—may be insufficiently potent to affect the relatively refractory adrenal of the patient with rheumatic fever.

There has been evidence to suggest that salicylates may cause an accelerated rate of "utilization" or metabolism of adrenal steroids.[9] In the light of these data, it seems possible that this effect may be exaggerated in the person with an active rheumatic inflammatory process, thus accounting for the failure of such individuals to develop elevated plasma concentrations of 17-OHCS in response to salicylate intoxication.

The observations of the present report are in conformity with those reported elsewhere that neither plasma concentrations[10, 11] nor urinary excretions[12, 13] of 17-OHCS are increased during salicylate therapy in patients with rheumatic fever. It has been stated[12] that the absence of an increased urinary excretion of 17-OHCS during salicylate therapy in rheumatic patients indicates that the therapeutic activity of salicylates in rheumatic diseases is not through stimulation of the pituitary and the adrenal cortex. This conclusion seems somewhat premature in view of the observed differences in response of rheumatic and nonrheumatic subjects to salicylate intoxication, and in view of the evidence, previously mentioned, that salicylates may cause an accelerated rate of removal of 17-OHCS from circulation by mechanisms other than urinary excretion. It seems possible that salicylates do stimulate the pituitary-adrenal system but that they may enhance the rate of removal of steroids from circulation sufficiently to prevent the demonstration of increased levels of plasma or urinary 17-OHCS.

SUMMARY

1. Effects of salicylate intoxication on plasma 17-hydroxycorticosteroid steroid concentrations have been studied in rheumatic and nonrheumatic subjects.

2. Patients with active rheumatic fever, in contrast to previously normal individuals, did not develop elevated plasma 17-hydroxycorticosteroid concentrations in response to salicylate intoxication.

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THE CAUSES OF INFANTILE CONVULSIONS PRIOR TO 1900

JOHN RENDLE-SHORT, M.R.C.P., D.C.H.
SHEFFIELD, ENGLAND

CONVULSIONS in infants are often dramatic, occur for no apparent reason, and are always potentially dangerous. It is probably for these reasons that they have fired the interest of medical observers all down the ages. On reading old medical textbooks it is noteworthy how frequently convulsions are mentioned. While this may be due partly to the striking nature of a fit, it does seem that, in fact, convulsions used to be more common in the past than they are today. Buchan,[1] for instance, stated in 1776 that "more children are said to die of convulsions than of any other disease"; and, as will be seen later, by the very nature of the conditions prevailing, fits must have been more common in previous centuries than they are in these more enlightened days.

SUGGESTED CAUSES OF INFANTILE CONVULSIONS

In the past, fits were commonly ascribed to teething. This belief seems to date from the time of the ancient Greeks. Hippocrates[2] (c. 440 B.C.) devoted one of his famous aphorisms (No. 25) to the subject of teething; this states:

> At the time of dentition there is pruritis of the gums, fever, convulsions, diarrhoea, especially when cutting the canine teeth and in those who are particularly fat and have constipated bowels.

Hippocrates is also credited by Still[3] with a short tract "On dentition." This frequently mentions fits, as in the following sentences:

> VI They that in teething have the belly much open are less convulsed than he that hath it seldom open.
>
> VII They that in teething have sharp fever come upon them, are seldom convulsed.
>
> X Not all that are convulsed whilst about teeth die, many come through it safely.

The first dentition was thought to be one of the main causes of convulsions until the beginning of the eighteenth century. Almost any writer on children's diseases could be quoted to illustrate this. Thomas Sydenham,[4] for instance, describes the benefits of bleeding children "during dentition, in the convulsions happening to them in the ninth or tenth month." But toward the end of the eighteenth century other causes were being suggested. Buchan[1] thought the reason so many children died of convulsions soon after birth was because they were often bound tightly round with five to six feet of swaddling clothes. He also quotes the case of a child who was found to have a pin sticking half an inch into its body "after it had died with convulsive fits, which in all probability proceeded from that cause." In his list of the causes of epilepsy Buchan includes: collections of water or serous humors on the brain, worms, teething, and "contagion received into the body" by smallpox, measles etc.

From the Department of Child Health, University of Sheffield.

A modern textbook of pediatrics, for instance Nelson's[5], lists about thirty causes of fits in children. Obviously these same causes prevailed in the past, though often unbeknown; but some which are rare today were common in the days when life was cheap, infections uncontrolled, and the necessity for vitamins not appreciated. A few of the more important causes will now be considered.

RICKETS

Livingston,[6] in his monograph on convulsive disorders in children, states that rickets was the main cause of convulsions in the past. According to Shankes[7] the first time that these two diseases were linked together was in a book by North[8] in 1826. When referring to "rachitis" he says, "Convulsions are a very frequent attendant of this distressing malady." North also described the syndrome we now know as spasmophilia—tetany, laryngismus stridulus, and convulsions, but he does not seem to have connected this condition with rickets. An earlier, according to Major[9] probably the earliest, account of spasmophilia had been given sixteen years before by Clarke[10] in 1815. He described dramatically the croup and carpopedal spasm, and pointed out that these were frequently the forerunners of a fit.

Many later writers have observed this combination of rickets, and spasmophilia. Henoch[11] wrote in 1889, "According to my experience the tendency to a convulsion is favoured by nothing so strongly as by rickets." He pointed out the frequency with which overt signs of rickets were found in children with convulsions and goes on to say, "Almost always with these attacks there are simultaneous attacks of laryngeal spasm." Gowers[12] in 1893 considered that the majority of so-called teething convulsions were in fact due to rickets, and the same suggestion was made by Hutchison[13] in 1905.

Shankes[14] showed that during the years 1922 to 1926, when rickets and tetany were rife, there was a well-marked seasonal incidence of convulsions with its maximum in the winter and spring. From this he concluded that the fits were related to rickets which had a similar seasonal incidence. During these years, too, the largest number of fits occurred in children between the ages of 6 and 12 months, which again is a common age for rickets. A comparable series of cases observed between 1942 and 1946, when, despite the war, rickets was a rarity, showed no such seasonal incidence and the majority of the cases occurred in infants under the age of 3 months.

Although rickets was first described by Whistler[15] in 1645 and was mentioned frequently in the London Bills of Mortality and in pediatric writings from then on, it is remarkable that the striking symptoms of spasmophilia achieved prominence only from about the beginning of the nineteenth century. If we assume that this means that it was then relatively a "new" disease, we would expect the incidence of convulsions to have risen at about the same time. In fact it seems to have done so. North[8] says that convulsions are "a much more frequent occurrence in the present day than at any former period." And, again, "in ancient Europe convulsive affections were scarcely known." He also records

that in 1792, of 17,650 children born in the Dublin Lying-in Hospital, 2,754 (about 15 per cent) *died* of convulsions during the first year of life. Doubtless in many hundreds more fits occurred but did not prove fatal.

The following facts therefore emerge: (1) toward the end of the eighteenth century there appears to have been an increase in the incidence of infantile convulsions, and (2) these convulsions were, apparently for the first time, frequently associated with tetany and rickets.

Rickets is due to lack of vitamin D, but spasmophilia is probably due to lack of calcium in the diet. These factors frequently overlap. We must now consider whether the calcium content of the diet of the people of this country was especially low during the nineteenth century. Drummond and Wilbraham,[16] in their masterly book *The Englishman's Food*, give a rough analysis of some diets from the fifteenth century onward. Table I is composed from their figures.

The drop in calcium content in the nineteenth century is most striking and reflects a change in the food habits of the people. Milk and milk products such as cheese are the main sources of calcium in human food, although eggs and flour also contain small quantities[17]; but milk does not keep well, especially when it is unpasteurized and not even clean. While England was an agricultural country this mattered little, but as the industrial revolution slowly engulfed the land, the difficulties of supplying the large towns with milk from the country became insuperable. For this reason milk in the big cities had to come from "stall fed cows kept in the dens and cellars of some of the wretched districts of London."[18] In 1865 the situation became even worse owing to an epidemic of cattle plague or rinderpest which destroyed large numbers of cattle.[16] It is not surprising that in 1826 North[8] was able to point out that the children of rich city dwellers were more prone to fits than the children of "those females who are regular in their mode of living and enjoy the calm tranquility of a country life." It is to be presumed that the children of these fortunate females enjoyed fresh milk in addition to the blessings of tranquility.

TABLE I*

	CALCIUM IN GM./DAY	VITAMIN D INTERNATIONAL UNITS/DAY
†Present-day diet	1.0	100?
15th century diet	1.2	?
17th century diet	1.5	?
Sailor's diet 1615	2.4	40
Diet St. Bartholomew's Hospital 1686	1.9	70
Navy ration 1745	1.0	15
Navy ration 1811	0.7	15
Gloucester goal 1815	0.5	0
Gloucester goal (when cheese allowed)	1.4	0
‡Dr. Kitchiner's estimate 1826	0.2	8
Sir J. Graham's prison diet 1843	0.4	0

*Data from Drummond and Wilbraham.
†For adult man engaged in light work.
‡Designed for artisan family in good circumstances.

BIRTH INJURY

The most common cause of convulsions during the neonatal period is birth injury. Today, with the virtual elimination of the maternal rachitic pelvis, with modern anesthesia permitting the easy application of forceps or other obstetric maneuvers and with cesarean section available if all else fails, the risk of

a prolonged labor involving great danger to the infant has been largely removed. But the obstetric art is young. Until the middle of the eighteenth century midwives were licensed by bishops, and in 1726 the Incorporation of Surgeons in Edinburgh acknowledge the great dangers "to women in childbirth and to their children through the ignorance and unskilfulness of midwives . . . who enter upon that difficult sphere at their own hand without the least trial taken of their knowledge of the principles upon which they are to practice that art."[19] The man-midwife at that time was often little better, and forceps were only just beginning to be used. William Smellie in his famous *"Treatise on the Theory and Practice of Midwifery"* records numerous case records of deliveries in which, for want of skill on the part of the attendants, dead or mutilated infants were brought into the world. He[20] records, for instance, that in the year 1746 he was called to a woman who, as the people about her alleged, had been in labor eight days; they said three midwives had attended and left her, that she was very poor and in a starving condition . . . "I found, by her own relations that the midwives had all tried to deliver her by hurrying and placing her in different positions, that she had got little or no sleep for two nights: that the waters came off the preceeding day and her pains had never since been stronger." Eventually Smellie delivered the woman of a dead baby. Such occurrences became less common as the teachings of this master obstetrician were disseminated, but the danger of cerebral injury must have been far greater in the past than it is now, and thus the incidence of infantile convulsions would be correspondingly higher.

INFECTIONS

Febrile convulsions may occur in infants at the onset of any feverish illness, such as an upper respiratory tract infection. But in addition to these comparatively mild infections, there are some diseases which are especially liable to be ushered in by a fit. According to Astruc,[21] "You rarely find a smallpox in children without epilepsy or convulsions." This deadly disease was rife in England before the introduction of vaccination by Jenner. Some idea of its prevalence can be gauged by the number of references to it in contemporary records. In the minutes of the London Foundling Hospital, measures taken to prevent the disease among the children and staff can frequently be found and include inoculation experiments carried out by Dr. William Cadogan.[22]

Probably both tuberculous and meningococcal meningitis were frequent visitors to the overcrowded hovels of the great cities. Fits often occur in these conditions. The following graphic description, although culled from a passage on the ubiquitous "teething," might well refer to meningococcal septicemia and meningitis:

> At the time of breeding the teeth, a fever often arises of the true inflammatory or effluent kind with great heat, high pulse, difficulty in breathing, a phrenzy of delirium, purple spots and petechial irruptions, convulsions, mortifications etc.[23]

PYRIDOXINE (VITAMIN B_6) DEFICIENCY

It is tempting to believe that pyridoxine deficiency may have been re

sponsible for some convulsions in the past. Long known to cause fits in experimental animals,[24] it has only recently been demonstrated that children can be affected in a similar fashion.[25] A pyridoxine-free milk was recently distributed in the United States which induced convulsions in many of the children who consumed it. The fits were controlled when pyridoxine was added to the diet or when the milk was changed,[26] but convulsions returned if the child was put back on the same diet.

It is possible that infants in the past could have been deficient in this essential factor. Vitamin B_6 is mainly found in seeds and cereals and especially in yeast. Milk contains 0.05, wheat flour 0.25, and whole wheat flour 0.5 mg. per 100 Gm. of food.[27] The minimum requirement for an infant is unknown but is probably about 0.15 mg. per day.[28] In the past, babies were fed on milk, usually from their mothers or a wet nurse, until about 9 or 10 months of age. They were then weaned onto pap; sometimes indeed they were fed on it from birth.[29] Many writers describe recipes for the preparation of pap. The following method described by Bonet[30] in 1684 can be taken as representative:

> Take barley or oatin-flower, put it in a bag of thick cloth, boil it in a kettle for twelve hours, so that the water may be always above it, keeping down the bag by laying a weight upon it. When the flower is taken out, set it in a hot oven, after the bread is taken out and keep the mass for use.

When required to feed the baby, this flour was mixed with water or boiled milk and given in a pap-boat.

It is little wonder that Conyers[31] warns against pap. He describes it as a sticky paste which would be more usefully employed by bookbinders for sticking pages together than as nourishment for infants.

It is difficult to say how much pyridoxine would remain in the pap after its preparation from flour. Vitamin B_6 is freely soluble in water and is readily hydrolyzed by weak acids.[27] It could easily have been lost in the water in which it was boiled. On the other hand, the majority of the vitamin is bound to protein which might save it from destruction.

There are other ways in which pyridoxine deficiency could have developed even though the diet apparently contained a sufficient amount of the vitamin. Some of the factors concerned have been listed by May.[32] They include inadequate stores passed to the fetus from the mother, infection, and inefficient synthesis in the intestine. The adequacy of this synthesis is probably related in some way as yet unknown to the composition of the diet.

Thus it will be seen that while pyridoxine deficiency could have been responsible for some convulsions in the past, it is difficult to be certain at this distance of time and without more exact knowledge of the diet.

LESS IMPORTANT CAUSES OF CONVULSIONS

Lead Poisoning.—Often the only symptom of lead poisoning in children is encephalopathy. This grave manifestation is ushered in by vomiting, irritability, and convulsions. In the past, lead pastes or lead nipple shields used by nursing women[33] have

caused poisoning in infants. In addition, the use of poor quality pewter with a high lead content for cooking and the storage of fruit and other food sometimes gave rise to lead poisoning in eighteenth century America.[34] Aub and associates[35] have shown that alcoholic beverages are especially liable to be contaminated by lead. In the eighteenth century and before, wine and spirits were often given to infants[36] and as children are more susceptible to lead than adults, this, too, may have been a source of poisoning.

Cysticerosis; Hydatid Disease.—It is interesting to speculate as to whether worm infestation could have caused fits. Certainly worms are frequently mentioned. In the period 1629 to 1660 the London Bills of Mortality recorded that 14,236 children died of "teeth and worms."[37] This figure is higher than for any other disease in the pediatric age group apart from "chrisoms and infants," 32,106. The high incidence of worm infestation does not cause surprise. Washing was an uncommon pasttime, garbage was thrown into the streets, and even medicines might contain filthy excrements as the following remedy for infantile convulsions shows:

> Take hens dung, dry it, part each piece in two and you will find a little white inside which you must take out dextrously with the point of a knife and beat it with loaf sugar and make thereof a powder. Give half a drachm thereof as you see occasions or a little more in broth or white wine.[38]

CONCLUSIONS AND SUMMARY

All the evidence and our natural expectations point to the fact that infantile convulsions were more prevalent in the past than they are today. The main cause in the nineteenth century was lack of vitamin D and calcium and there was a close association with rickets. In previous centuries birth injury, infection both intracranial and extracranial, and, possibly to a lesser extent, lead poisoning, cysticerosis, and hydatid disease can be incriminated. Whether pyridoxine insufficiency played any part it is impossible to say.

It is a pleasure to record the help received from the Librarians of the University of Sheffield, the Royal College of Physicians, London, and the British Medical Association.

My thanks are also due to Prof. R. S. Illingworth for his helpful criticism.

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SEPTICEMIA AND MENINGITIS IN A NEWBORN INFANT

Report of an Unusual Example Due to Simultaneous Infection by Escherichia coli and Pseudomonas aeruginosa, With Recovery

Richard T. Smith, M.D.
Minneapolis, Minn.

IN SPITE of the great progress in the control of infection in newborn infants, deaths continue to occur as a result of septicemia and meningitis in the neonatal period. Indeed, according to several recent studies, septicemia of the newborn still carries a high mortality rate, particularly when associated with meningitis.[1, 2, 3]

Several cases of meningits due to simultaneous infection by two distinct bacterial species have been reported. In only two instances have the organisms both been of enteric type.[4] This report describes the successful treatment of a premature infant with meningitis and septicemia due to both *Escherichia coli* and *Pseudomonas aeruginosa*, complicated by meningitis, multiple abscesses, scleredema, and erythema multiforme. This unusual case is reported because it re-emphasizes the occurrence, the clinical nature, and the importance of appropriate intensive treatment of major infections due to enteric microorganisms in the newborn period.

From the University of Minnesota School of Medicine, Department of Pediatrics, and the Research Laboratories of the Variety Club Heart Hospital.

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CASE REPORT*

R. K., a white male infant, the second child of a 22-year-old, healthy housewife, was referred to the University of Minnesota Hospitals at the age of 3½ days with a history of vomiting for forty-eight hours, jaundice and cyanosis for twenty-four hours.

The mother's pregnancy terminated at thirty-three weeks, when after four days of intermittent labor and artificial rupture of the membranes, delivery followed spontaneously within twenty-four hours. There was no evidence of intrapartal or postpartal infection noted in the mother.

At birth the child weighed 2,400 grams, breathed spontaneously, and exhibited normal color and cry. The neonatal course was uneventful until 24 hours of age, when he vomited the first feeding offered, and had the first of several episodes characterized by the development of a dusky gray color and tachypnea. On the second day of life he began to appear jaundiced, increasing in intensity until admission to the hospital.

Physical examination on admission to the University Hospitals revealed a gravely ill immature infant in severe distress. The weight was 2,420 grams, length 46 centimeters, temperature 96° F., pulse 200, respiratory rate 50. His general appearance and the feel of the tissues to palpation were singular. The infant appeared grossly edematous, including extremities, serotum and face. The skin was a dusky vermilion color which, when pressed

paled, revealing an underlying ieteric tint. To the palpating finger, the skin, the subcutaneous tissues, and the muscle gave a cool, brawny, nonyielding cadaverous sensation. This was particularly marked on the thighs, abdomen, and face, although the texture was uniform, not spotty, in distribution. There was no skin rash at that time. The fontanel was soft and depressed. The eyelids were very edematous; however, it could be seen that deep reflexes could not be elicited, the Moro reflex was weak, and the cry was high pitched and plaintive. There was no detectable lymphadenopathy.

Initial laboratory work revealed the hemoglobin to be 16.6 grams per 100 ml.; white blood cell count was 2,100 per cubic millimeter with 2 per cent mature neutrophils, 55 per cent immature granuloeytes, 27 per cent lymphocytes, and 11 per cent macrophages or histiocytes. Roentgenograms

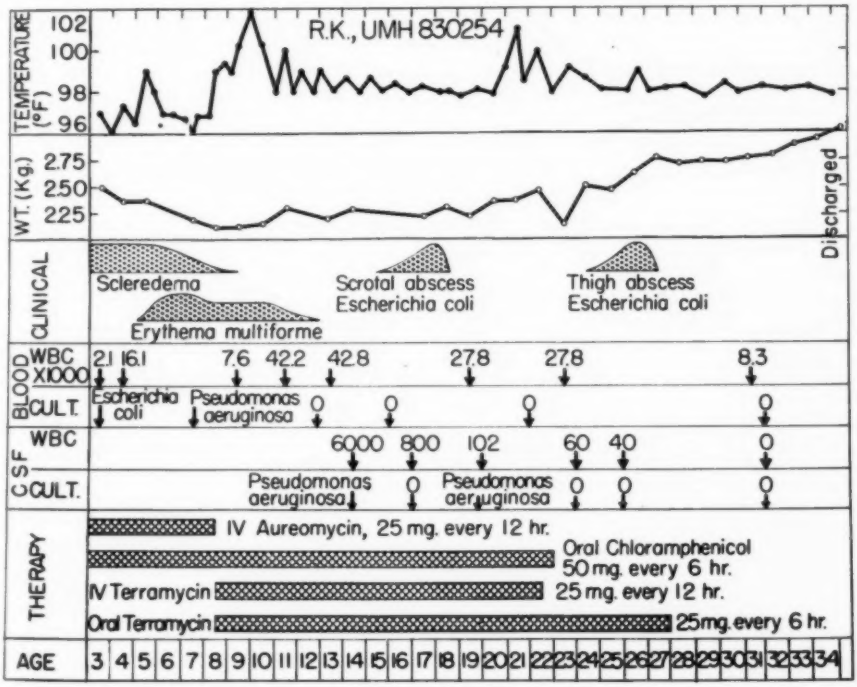

Fig. 1.

the pupils reacted sluggishly to light and that the scleras were ieteric. The chest was clear; the heart normal in rate, rhythm, and quality of heart sounds. The abdomen was slightly distended, tense, and tympanitic. The umbilicus was dry and healing. The liver edge was felt 2 cm. below the right costal margin; the spleen was not enlarged. Movements of the extremities were markedly limited, especially at the knee and hip. The of the chest and abdomen were normal. The blood urea nitrogen was 25 mg. per cent, the CO_2 combining power 18 meq. per liter, the serum chlorides 110 meq. per liter, and the serum sodium 157 meq. per liter. Urinalysis showed a trace of albumin, no cellular elements, and a negative test for bile. The child was type O, Rh negative, and the Coombs test was negative. The precipitin test for C-reactive protein was strongly positive.

After obtaining blood and throat cultures, specific antibiotic therapy was begun with chlortetracycline, intravenously, 15 mg. every 12 hours, and chloramphenicol orally, 50 mg. every 6 hours. Fig. 1 shows the temporal relationships of the various clinical and laboratory features of this infant's illness as related to therapy.

Culture of the initial blood sample showed 148 colonies per cubic centimeter of *E. coli* after twenty-four hours' incubation. This organism was found subsequently to be sensitive to 62.5 μg per milliliter of streptomycin, 7.8 μg per milliliter of chlortetracycline, 1.9 μg per milliliter of oxytetracycline, and 3.9 μg per milliliter of chloramphenicol. The throat culture taken at the time of admission revealed *Ps. aeruginosa*. After therapy was initiated, the browny edema and icterus rapidly subsided, and during the next forty-eight hours the infant's tissues gradually acquired a normal pink color and soft velvety texture. The body temperature, however, failed to stabilize and the infant continued to be very listless.

On the fifth day of life, the day following disappearance of edema, an erythematous macular rash made its appearance. Large, red, raised areas on the skin spread centrifugally, clearing and blanching in the center, the margins remaining red and discrete. These lesions coalesced in some areas over the thighs and abdomen, and after three days disappeared entirely. At the height of the rash, the white blood cell count had risen to 16,000 per cubic millimeter with only 2 per cent histiocytes, 68 per cent immature granulocytes, 2 per cent mature granulocytes, 22 per cent lymphocytes, and 6 per cent eosinophils. *Ps. aeruginosa*, 5 colonies per milliliter, grew out in a second culture of the blood after forty-eight hours' incubation. This organism was found to be sensitive to 31.2 μg per milliliter of chlortetracycline, 3.9 μg per milliliter of oxytetracycline, but was insensitive to chloramphenicol, and to a combination of chloramphenicol and penicillin or streptomycin.

The infant continued to feed poorly, was listless, weak, and inactive. No bulging of the fontanel, stiffness of the back or neck was detected at any time. On the fourteenth day of life a lumbar puncture yielded a yellowish, turbid fluid under normal pressure, containing 6,000 white cells per cubic millimeter, all polymorphonuclear leukocytes. The sugar level was less than 15 mg. per cent and smears revealed large numbers of fat, gram-negative rods, mostly within cells. *Ps. aeruginosa* grew out in pure culture.

Progress following this was satisfactory. All subsequent blood cultures were sterile. The spinal fluid cell count decreased; however, the sugar remained low, and a culture on the eighteenth day of life grew out *Ps. aeruginosa*. On the sixteenth day of life a firm mass appeared in the serotal sac which soon became fluctuant. Incision of the mass yielded yellow pus which grew *E. coli* in pure culture. After the eighteenth day of life the spinal fluid showed a rapid decrease in cell count, increase in sugar, and two successive sterile cultures. Oral and intravenous oxytetracycline and oral chloramphenicol were continued, however, through the twenty-second day of life.

On the twenty-first day, the temperature rose to 101° F., and gradually subsided over a three-day period; the peripheral leukocyte count remained elevated. A second abscess appeared in the right anterior thigh which likewise yielded, upon incision, yellow pus containing *E. coli*. On the twenty-seventh day all antibiotic therapy was stopped. Blood studies were normal after this; blood and spinal fluid cultures remained sterile. The child began to gain weight, became alert and active.

The urinalysis had been consistently normal, but investigation for a portal of infection prompted culture of the urine. These cultures grew out *E. coli* on three occasions during convalescence and after stopping all anti-

biotic therapy; subsequent to this, two more cultures were sterile. An intravenous pyelogram performed before discharge was not entirely satisfactory, but demonstrated normal position of the kidneys bilaterally, adequate function, and no gross calyceal distortion.

Following discharge the child did well, eating and gaining weight in a normal fashion. At 6 months of age he was able to follow, laugh in response to his mother, reach and grasp, and showed at that time no pathological neurological signs. At the age of 2½ years, a thorough examination showed a talkative boy with no complaints. Head size, neurological examination, and intelligence all were found to be within normal limits. No liver enlargement or other physical abnormalities were found. Blood count and urinalysis were negative.

DISCUSSION

A premature newborn infant developed on the second day of life a progressive illness characterized clinically by vomiting, jaundice, cyanosis, respiratory difficulty, scleredema, and, after the beginning of therapy, an erythema multiforme rash. Laboratory examinations revealed leukopenia, histiocytosis, and *E. coli* in cultures of the blood and of two metastatic abscesses. *Ps. aeruginosa* was isolated from a subsequent culture of the blood. After treatment had been under way for ten days, purulent meningitis was uncovered, with growth of *Ps. aeruginosa* on culture. Treatment with intravenous chlortetracycline and oxytetracycline, and oral oxytetracycline and chloramphenicol over a twenty-three-day period resulted in eradication of the infection and a healthy child.

Review of the available literature fails to disclose a similar case of meningitis and septicemia due to enteric microorganisms. In most instances of double infections on record, the combination has been an enteric microorganism with a gram-positive one, such as a pneumococcus, staphylococcus, or streptococcus.[5-9] Kagan and associates,[4] in reviewing a twenty-year experience, found twelve cases of meningitis in 7,129 premature infants seen at the Sarah Morris Hospital. In ten of those patients, the infection was due to enteric microorganisms. In three, mixed infection was found: *E. coli* and *Klebsiella pneumoniae, E. coli* and *Aerobacter aerogenes,* and *E. coli* and a pneumococcus. Blood cultures were not reported. All but one infant in their series died.

It seems probable that the infant reported here had meningitis for ten days during intensive antibiotic therapy before its presence was suspected and a lumbar puncture performed. Failure to do well in regard to temperature regulation, feeding behavior, and weight gain prompted this procedure; there was no change in head size, fontanel, no seizures, and no stiffness of the neck or back. These findings are in accordance with the literature where it has been stressed that absence of the usual meningeal signs in the newborn and particularly the premature infant is to be expected in early meningitis due to any microorganism.[1, 4, 10] However, meningitis of the duration assumed in this case usually gives evidence such as bulging fontanel, enlarging head, convulsions, or neurological signs. Antibiotic therapy directed toward microorganisms circulating in the blood apparently suppressed the usual manifestations of meningitis.

Leukopenia and histiocytosis were outstanding early laboratory features

in this patient. It has been pointed out that overwhelming infection in the newborn period may be associated with normal or low peripheral white blood counts[3, 5]; however, the possible relationship of such hematological findings to the type of infecting microorganism has not received emphasis. Leukopenia is a well-recognized response to circulating gram-negative microorganisms or their endotoxins at any age, for example, the reaction to contaminated parenteral fluids, or typhoid vaccine given intravenously. It is interesting in view of the large numbers of gram-negative bacteria circulating in the infant's blood to speculate that such a mechanism might be operating in this patient, resulting in leukopenia. The presence of macrophages in the circulating blood of a newborn infant with septicemia has not previously been reported.

It has been well established[10-14] that most examples of meningitis occurring in the newborn period are due to one of the microorganisms of the enteric group. No adequate explanation of this interesting and unique association has been proposed. The pathogenesis of meningitis in the newborn period is likewise poorly understood. It seems probable, however, that meningitis during this period of life, in the absence of congenital defects or trauma, represents a sequela of bacteremia. Demonstration of bacteremia in a large proportion of the reported cases of meningitis during the newborn period in which the blood has been cultured[1, 3, 4, 10, 13] adds considerable weight to this point of view.

Successful therapy in the presence of severe and overwhelming infection in the newborn infant depends upon the early and intensive use of antibiotic agents active against the enteric group of microorganisms usually responsible for these infections.[1] It has been shown that penicillin alone or in combination with streptomycin is not adequate in septicemia and meningitis due to enteric microorganisms. Twenty-one of twenty-five infants treated with this combination in one series[6] failed to survive. Recent case reports and one larger series[1] have indicated a more favorable response of these severe infections to chlortetracycline, oxytetracycline, chloramphenicol, polymyxin, or neomycin. There is no evidence to indicate which of the broad spectrum antibiotics now available might be best.

SUMMARY

A case of septicemia and meningitis in a newborn premature infant due to *E. coli* and *Ps. aeruginosa*, and associated with scleredema, erythema multiforme, leukopenia, and histiocytosis, is reported. Therapy with broad spectrum antibiotics resulted in complete recovery. The etiology, pathogenesis, and treatment of meningitis in the newborn infant are reviewed briefly.

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June 3 1752. This morning my Cousin Benj: Loring of Hingham call'd at my house by whom I was informed that there had been of late a great mortality both at Hingham and Weymouth. At Hingham 80 persons (if I mistake not) have Died of the Throat Distemper beginning in October but raging most in the winter. At Weymouth an 100 by the same terrible Disease. And since have Lost most of their principal men by Pleuritic fever. The Lord sanctify to his people in those towns, such an awful visitation of his providence, and teach them to profit thereby, And teach me, o Lord, to make a right improvement of it, And when thy Judgments are abroad in the earth may the Inhabitants thereof learn righteousness therby.

June 29. This day Died a daughter of Nathan Goodenow of the throat distemper. 'Tis not long since sorrowful breaches were made in several families among us by this mortal distemper.

DIARY OF REV. ISRAEL LORING OF SUDBURY.

ENTEROCOCCUS PNEUMONIA AND SEPTICEMIA IN A TWO-MONTH-OLD INFANT

WALTER LEVY, M.D., AND ELISABETH TURNAUER, M.D.
NEW YORK, N. Y.

ENTEROCOCCUS infections are not uncommon in the genitourinary tract. They are less frequently observed in the gall bladder, the gastrointestinal system, the endocardium, and the central nervous system.[1-3] Rarely are they encountered in the lungs. All reported cases of enterococcus pneumonia that we have found in the literature have been noted in adult patients.[1, 3-6] Some of these have had enterococcus septicemia as a complication.[3, 5, 7] The clinical course of enterococcus pneumonia and septicemia has been characterized by relative chronicity with exacerbations, occasional pleuritis, lung infiltrations, and cough with fetid and sometimes bloody sputum.[1]

Recently, we had the opportunity of observing a case of enterococcus pneumonia with a complicating septicemia in a 2-month-old infant. The pediatric literature available to us does not disclose a pneumonic syndrome associated with this organism.

CASE REPORT

A. B., a 2-month-old white male, was admitted to Morrisania City Hospital on Feb. 18, 1954, with a history of cough for three days, fever for two days, and difficulty in breathing for about eighteen hours. One week earlier the child had been seen and treated for a cold, with Gantrisin, in the outpatient department. The dosage given was 0.5 Gm. three times daily. Past history and family history were noncontributory.

Fig. 1.—Feb. 18, 1954. Showing increased density over the entire right lung field with several round areas of decreased density in this region. Fluid level is seen at the right base.

The temperature was 100.6° F. Physical examination revealed mild pharyngeal injection, flatness to percussion over the entire right side of the chest with crepitant râles audible over the same area, and a few crepitant râles over the left pulmonary field.

X-ray examination disclosed increased density over the entire right lung field. Several round areas of decreased density were present in this region. A fluid level was seen at the right base. Blood count showed a hemoglobin of 9.0 grams, red cells 3,100,000, white count 8,100 with 6[illegible]

From the Pediatric Service of the Morrisania City Hospital (Dr. Harry D. Pasachoff, Director) and the New York Medical College.

per cent polymorphonuclear neutrophils and 31 per cent lymphocytes. A growth of enterococcus was recovered from throat and blood cultures. The organism was found to be sensitive to Terramycin, Ilotycin, Chloromycetin, Aureomycin, bacitracin, and resistant to penicillin and streptomycin. Urinalysis was not remarkable.

The child was placed in an oxygen tent. Initial therapy was Terramycin 125 mg. administered every six hours orally, syrup of Gantrisin 0.25 Gm. every six hours orally, and penicillin intramuscularly as follows: crystalline penicillin 200,000 units every three hours for eight doses and procaine penicillin 300,000 units twice daily. Despite oxygen administration the child continued to be dyspneic. Dullness to percussion, decreased breath sounds, and numerous moist râles over the right lung field persisted. Two days later the temperature was 102.6° F., but returned to normal on the fourth hospital day. Blood culture and sensitivity tests became available five days after admission, and penicillin and Gantrisin were then discontinued. The child continued to receive Terramycin 125 mg. every six hours and Ilotycin 50 mg. three times daily by mouth.

X-ray examination on the sixth day disclosed extensive cavitation. The hemogram on that day revealed more marked anemia with a hemoglobin of 8.0 grams and an increase in the white blood cell count to 20,300. A thoracentesis of the right pleural space was performed because of persistent respiratory distress. Three cubic centimeters of viscid, blood-tinged fluid was obtained from the eighth intercostal space. The ninth intercostal space was then aspirated and 0.5 c.c. of thick, purulent, greenish material was removed. This substance was smeared and gram-positive cocci in short chains were isolated. However, no growth was obtained on culture. A blood culture on the same day was also sterile. Chest x-ray on the following day showed an increase in the size of the abscess cavity in the right mid-lung field. The remainder of the right lung was homogeneously dense.

Fig. 2.—March 9, 1954. Reveals increase in size of the large cavity in the right lower lung field.

Fig. 3.—Jan. 4, 1955. Showing no evidence of pneumatocele or emphysema in the right lung. Enlargement of right paratracheal lymph nodes is noted.

The clinical findings remained unchanged until March 1, 1954, when it was noted that the child appeared improved, was more alert, and was taking feedings well. X-ray revealed the abscess cavity to involve the entire lower right lung field. However, clinical improvement continued and on March 6, 1954, the child was taken out of oxygen. A subsequent roentgenogram revealed an increase in the size of the abscess cavity, but slight resolution of the density in the right upper lung field. Terramycin was discontinued on March 11, 1954, on the twenty-first hospital day. Shortly

thereafter the physical signs of dullness and decreased breath sounds were less obvious. Coincident with this the hemoglobin rose to 11.5 grams and the white cell count dropped to 16,150. The differential count was 28 per cent polymorphonuclear neutrophils, 70 per cent lymphocytes, and 2 per cent monocytes. Ilotycin was discontinued on the thirty-sixth hospital day. Six weeks after admission, x-ray of the chest revealed considerable diminution in the size of the cavities of the right lung with slight resolution of the pneumonic process. The child was discharged on the forty-fifth day.

The patient has been followed at monthly intervals. He has gained weight, developed normally, and is doing well. There are no abnormal physical findings. Serial x-rays revealed a gradual resolution of the pneumonic process in the right lung. Since Oct. 5, 1954, eight months after the onset of the illness, no pathology of lung tissue has been evident on x-ray examination. There is a residual enlargement of the right paratracheal nodes which has persisted until the present time.

LABORATORY EXAMINATION FOR IDENTIFICATION OF THE ORGANISM

One bottle of thioglycollate and one bottle of soy trypticase broth, each containing about 100 c.c. of medium, were used for blood cultures. Blood was obtained under sterile precautions and approximately 5 c.c. of blood was placed in each bottle. The blood cultures were incubated at 37° C. and subcultured at regular intervals. Gram-positive cocci in pairs and in short chains were isolated from the blood and throat cultures. The microorganism was identified as enterococcus, and exhibited the following characteristics: growth on blood agar produced a green zone around the colony; viable organisms were present after exposure to 60° C. for thirty minutes; growth occurred in 6.5 per cent saline broth; the direct Neufeld test was negative, and no solubility in bile was observed.

BACTERIOLOGY

Several types of streptococci have been classified as enterococci because they share certain common characteristics.[2] All are in the Lancefield group D. Enterococci are divided into four large groups: *Streptococcus fecalis*, *Streptococcus liquefaciens*, *Streptococcus durans*, and *Streptococcus zymogenes*. Morphologically they are gram-positive cocci found in pairs and occasionally in short chains.

The cultural characteristics reveal them to be resistant to heating at 60° C. for thirty minutes; they grow in 0.1 per cent methylene blue in milk, and remain viable in low temperatures (10° C.) as well as in high temperatures (45° C.). Growth is seen in broth containing 6.5 per cent sodium chloride, and in broth adjusted to a pH of 9.6; they also grow on 10 and 40 per cent bile blood agar. The final pH in 1 per cent glucose broth is 4.0 to 4.5.

Enterococci may be alpha, beta, or gamma streptococci, and therefore may or may not cause hemolysis on blood agar. The morphological characteristics of the colonies depend on the type of enterococcus.

Enterococci have been isolated as normal inhabitants of the mouth and nasopharynx.[1] Nevertheless, they may be true pathogens. Strains obtained from healthy individuals have not produced pathological lesions when injected into laboratory animals, whereas cultures secured from sick patients have been found to be pathogenic.[8]

COMMENT

This infant was critically ill for ten days despite the administration of antibioties. Nevertheless, even though clinical improvement was obvious, x-ray evidence of cavitation and pneumonic infiltration persisted for eight months before complete resolution could be observed. Therapy was continued for six weeks in order to prevent a relapse. The isolation of enterococcus from the nasopharynx offers a plausible explanation for the portal of entry of this organism. The meninges, genitourinary tract, and the endocardium were not involved in our patient. These systems are more frequently the sites of pathology in adult patients.[1, 2]

The paucity of enterococcus pneumonias is remarkable since the organism is frequently encountered as a normal inhabitant of the nasopharynx and gastrointestinal tract. We are unable to explain why these bacteria should spontaneously change from nonpathogens to those of such virulence. Neither has the chronicity of the pulmonary lesions been adequately explained.

The enterococcus is ordinarly sensitive to Terramycin, Aureomycin, Tetracycline, Chloromycetin, and Ilotycin, and resistant to penicillin, streptomycin, and sulfonamides. In our case, the organism followed the usual pattern of sensitivity and resistance. Terramycin and Ilotycin were very effective.

SUMMARY

Enterococcus pneumonia associated with septicemia, pleuritic involvement, and cavitation was observed in a 2-month-old infant. The blood stream infection was transient. However, the pulmonary pathology persisted for eight months before complete resolution could be detected. Therapy with Terramycin and Ilotycin was very effective in controlling the disease.

We wish to acknowledge the assistance of Miss Dorothea Richtberg, Bacteriologist of the Morrisania City Hospital.

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THE EFFECT OF CERTAIN TYPES OF TRAUMA IN EPIDERMOLYSIS BULLOSA

CLAUDIA R. SIMON, M.D.
CINCINNATI, OHIO

THE differential diagnosis of bullous disorders in infants and children is often difficult. Any diagnostic aids which may help should be of considerable interest. A study of the various types of trauma able to evoke a bullous reaction in epidermolysis bullosa has been made.

Little is known about the mechanism of bullae formation in epidermolysis bullosa. Engman and Mook[1] were the first to believe that a developmental defect of elastic tissue is present. But this has not been confirmed by other investigators.[2, 3]

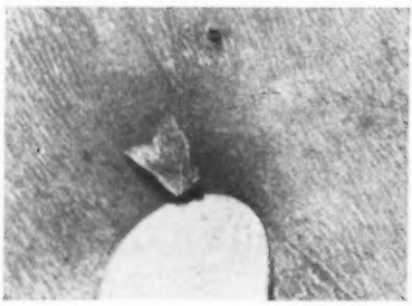

Fig. 1.—Dystrophic epidermolysis bullosa. Epidermolysis produced by rubbing with tongue blade.

The following is a description of various types of trauma which will produce lesions in a patient with epidermolysis bullosa, and again demonstrates a lack of cohesive force of the skin in this disease.

From the Department of Dermatology and Syphilology, College of Medicine, University of Cincinnati.

CASE REPORT

The patient was K. W., a one-year-old white female, with typical lesions of the dystrophic type of epidermolysis bullosa, present since birth. The patient had generalized lesions involving skin and mucous membranes and presented serous and hemorrhagic bullae, denuded and crusted lesions, scarring, and nail involvement. She was treated with pregnenolone 100 mg. every day, which resulted in some improvement.

Fig. 2.—Epidermolysis produced most constantly by adhesive application.

The following types of trauma were applied to skin areas of the back, which at the time were free from visible lesions. The patient did not receive any therapy during the performance of this procedure.

1. Multiple light tapping with a percussion hammer produced immediate, severe, and prolonged erythema. If the tapping was continued, denudation of the skin occurred.

2. Scraping with a tongue blade also produced denudation of the skin (Fig. 1).

3. Application of adhesive tape resulted in a stripping off of the top layers of the skin, so that they adhered to the tape when it was removed (Fig. 2). This procedure was the most constant, as it could be reproduced over nearly the entire skin surface.

4. Multiple pinpricks produced minimal erythema, but no other lesions.

5. Intracutaneous injection of 0.02 c.c. of hyaluronidase produced no abnormal reaction in the skin. A control injection of 0.02 c.c. of saline also did not produce any lesions.

Leoni[4] has made a somewhat similar study in epidermolysis bullosa simplex and concluded that the primary disturbance in this disease resulted from a defect in elastic tissue development and possibly a hypersensitivity of cutaneous capillaries.

These techniques of a trauma test were applied to two other cases of epidermolysis bullosa with similar results, to clear skin of an infant with Ritter's disease with negative results, to generalized impetigo in two young children with negative results, and also to a case of severe poison ivy dermatitis with equally negative results.

CONCLUSIONS

Trauma from the pulling up of a strip of adhesive tape has been found to produce uniformly epidermolysis in two cases of epidermolysis bullosa. This simple trauma test may be of help in the differential diagnosis of bullous diseases of the skin in infants and children.

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THE TREATMENT OF ECZEMA IN INFANTS AND CHILDREN

PART V

LEWIS WEBB HILL, M.D.
BOSTON, MASS.

ATOPIC DERMATITIS—CONT'D

ENVIRONMENTAL ALLERGENS

The environmental allergens which most frequently cause atopic dermatitis are house dust, pollen, and wool. In addition to these I have seen it caused by horse and cat dander, cottonseed, Kapok, and feathers. It is probable that these allergens, with the exception of wool, work by inhalation more than they do by contact.

For young infants, house dust and wool are the most important of the environmental allergens. Pollen as a cause of atopic dermatitis is unusual in infants—the youngest child that I have seen, in whom I was sure it was a cause, was 2 years old. If my figures for intracutaneous skin tests with dust and feathers mean anything, and I am not entirely sure that they do, these two allergens are frequent causes of atopic dermatitis in infants; this is, however, pretty hard to prove. As a routine treatment it is well to remove feathers from the environment, and dust and wool in so far as it can be done. What one sees so often is a highly allergic baby with atopic dermatitis in whom it is difficult to prove any specific clinical sensitivity except for egg white.

It is in children after the age of 2 years that the environmental allergens are most important, and if the eczema began late, that is, after the age of 10 or 12 months, and particularly if it began back of the knees and in front of the elbows instead of on the face, the chances are that it is due to environmental allergens. I am sure that many children after the age of 2 years have atopic dermatitis from house dust.

In children who have had atopic dermatitis for several years, a history of seasonal exacerbation is of the utmost importance. Many of these children are worse in the winter and their eczema begins each fall, "as soon as the heat is turned on." It lasts all winter, reaches its height in March or April, and then begins to subside, so that during the months of June, July, and August there is little or no eczema. Such eczema is often due to wool or to house dust or to both.

Another group of patients is nearly free from eczema in the winter, and has it especially during the pollen season, with or without hay fever. These patients are common, and it seems clear that their eczema is due to pollen. This is probably caused by inhalation of pollen, and not by contact (see Contact Dermatitis), for scratch tests of the urticarial type are positive, and patch tests with whole

Editor's Note: Despite the notice in the August issue that reprints would not be available of Dr. Hill's series of papers on "The Treatment of Eczema in Infants and Children," many requests are being received. As previously stated, no reprints are available as the articles will be published in book form.

pollen or with pollen oil, at least in my experience, which is only with children, are negative. Still another group of children has atopic dermatitis all the year round. Sometimes these children are sensitive to dust and wool, which accounts for their winter trouble, and also to pollen, which accounts for their summer trouble, and some may be sensitized to still other environmental allergens such as feathers and animal dander. I have seen a boy of 7 years, whose eczema was caused solely and entirely by feathers. He lived on a chicken farm where there were 2,000 chickens.

Food is sometimes a partial cause of atopic dermatitis in older children but I think environmental allergens are of far more importance. This has been emphasized especially in the last few years by Louis Tuft of Philadelphia, and he has quite definitely caused exacerbations of atopic dermatitis by spraying house dust into the nose of one patient and ragweed into the nose of another. I have seen much atopic dermatitis due to wool, although I am not quite so enthusiastic about it as is Dr. Earl Osborne[21] of Buffalo, who has studied this subject for years. I think that wool works more often by contact than it does by inhalation. Dr. Osborne has pointed out, however, that what we call "house dust" is to a large extent made up of wool fiber, and he is convinced that it works as frequently by inhalation as it does by contact. However, scratch tests to wool are uncommon, and when they do occur, the degree of sensitization is low. Wool is a weak allergen, although rarely the degree of sensitivity is so great that simple contact of wool with the skin will cause urticaria, and I have seen one child who wheezed if a wool blanket came near his nose. Such situations are, however, unusual. In 200 cases I found only one positive scratch test. Dust, however, in older children with atopic dermatitis gives a high percentage of positive scratch tests (I have no accurate figures) and many of these are strong reactions. Furthermore, it has been clearly shown that while dust is made up of all sorts of small particles, and that a good many of these are wool particles, the allergenic activity of dust depends not so much upon these as upon its own specific allergen.

Children with wool sensitivity are likely to have eczema around the neck, on the wrists and the back of the hands (mittens), and around the ankles, if they have been wearing a snowsuit, and the legs get wet, as they usually do. There is also likely to be eczema in front of the elbows and back of the knees which is probably caused by absorption of the allergen from other localities or possibly by inhalation. Wool is also irritating to many skins entirely apart from any question of allergic sensitization.

In a recent series of forty cases of "winter" atopic dermatitis in children between the ages of 2 and 12 years I found no positive scratch tests to wool, ten positive intracutaneous tests, and fourteen positive patch tests. There was no correlation between the positive patch and intracutaneous tests—either might occur without the other. A positive patch test* is pretty good

[21]Osborne, Earl D., and Murray, Philip F.: Atopic Dermatitis. A Study of Its Natural Course, and of Wool as a Dominant Allergenic Factor, Arch. Dermat. & Syph. 68: 619, 1953.

*The wool patch should be left on for five days. The sensitivity is of low degree, and it is rare for a positive test to show if the patch is removed in twenty-four hours.

evidence that the eczema is being caused by wool, a positive intracutaneous test probably means very little one way or the other. A negative patch test, on the other hand, by no means excludes wool as a cause of the eczema, because in the patch test the element of rubbing is not present, which is probably of considerable importance in causing the eczema.

It is not easy to avoid wool. What I have usually done is to have the children wear Byrd cloth or nylon snowsuits, leather mittens lined with cotton, cotton or nylon jerseys, remove wool rugs from their bedrooms, and either get rid of their wool blankets or have them enfolded in cotton sheets.

Fig. 24.—Atopic dermatitis from wool. Easily treated because it can be bandaged. Tar paste is indicated, or Swartz's ointment made without lanolin.

HYPOSENSITIZATION TO ENVIRONMENTAL ALLERGENS

It is not possible to avoid some environmental allergens, so that the question of hyposensitization naturally suggests itself. Walker[22] in 1918 was the first to discuss this. He said: "Eczematous patients tolerate very small doses of the offending protein and the eczema seems to improve; but a slight increase makes the eczema worse. The amount of protein that benefits eczema is too small to prevent asthma, and the amount that benefits asthma makes the associated eczema worse. Desensitization for eczema must be a very slow and cautious process." This was written thirty-seven years ago. It was true then; it is true now.

There is, however, considerable difference of opinion as to the value of hyposensitization. Some place it on a par with hyposensitization in hay fever; some believe that it is worthless. All are agreed with Walker's original statement that very small

[22]Walker, I. C.: Causation of Eczema, Urticaria and Angioneurotic Oedema by Proteins Other Than Those Derived From Foods, J. A. M. A. 70: 897, 1918.

doses of antigen must be used if exacerbations of the dermatitis are to be avoided. A severe exacerbation is no light matter, and it may be a good while before it calms down.

It has seemed to me that while hyposensitization is sometimes worth while, it can be by no means put upon the same plane as hyposensitization in hay fever. The situation is quite different. In hay fever the symptoms are brought about by local contact of pollen with the nasal mucosa; in atopic dermatitis due to pollen the pollen antigen has been absorbed and brings about its effects from within. If it is admitted that circulating pollen antigen causes the trouble, it is not logical to expect good results by adding more antigen to that which is already present. For this reason I am somewhat doubtful of the beneficial effects of such inoculations while the patient is still in his dusty house or during the pollen season. However, these are all obscure matters, with many questions unanswered, and I am sure that hyposensitization may sometimes do good. I have seen some patients made much worse by the injection of only a very small dose; I have seen some for whom attempted hyposensitization did no harm or no good; and I have seen a few where it was undoubtedly of real benefit. For pollen dermatitis it is the only thing that *can* be done, except local treatment or treatment with cortisone. In the last two years I have given the inoculations intracutaneously rather than subcutaneously. Whether this is really any better than the subcutaneous method, I do not know. My recent practice has been to give once a week (later every two weeks) just enough extract (dust or pollen) to produce a reaction about as big as a nickel. In two patients being treated now this is brought about by the injection of about 0.05 c.c. of a 1 to 10,000 pollen extract. The dose is not increased—no attempt is made to attain a maximum tolerated dose as there is in treating hay fever; if you keep raising the dose you will almost certainly reach the point where there will be an exacerbation of the eczema. I have taught one mother to give these intracutaneous injections—the dose is so small that there is no possibility of a general shock reaction.

Of the children with atopic dermatitis from wool whom I have seen, I have used hyposensitization in only three. These patients had such severe eczema that it was incapacitating. The dosage of wool extract used was 0.20 to 0.40 c.c. of a 1 to 10,000 dilution given subcutaneously. One of these patients had a severe flare-up after a dose of 0.05 c.c. of a 1 to 1,000 dilution. It seemed to me, and to the patients, that hyposensitizing inoculations, which were done for several years in each case, were of definite benefit. The inoculations were started in September, carried through the winter, and discontinued in the summer.

PSYCHIATRY

There can be no doubt that emotional disturbances are of great importance in aggravating atopic dermatitis. That they are ever a primary cause, I do not believe, and I cannot agree with those who think that psychiatric treatment is the most important thing in dealing with these patients. Like all good things, the psychiatric aspects of atopic derma-

titis have been overemphasized. The older the patient, the more important psychiatry is; I am very doubtful that it has much to do with atopic dermatitis in babies, in spite of what some enthusiasts have said, except that when they are overtired or overexcited they scratch more. There can be no doubt, however, that in older children mental tension of any sort can aggravate the eczema. This is particularly true of girls and boys in high school or college who are conscientious about their studies and are having difficulty with them—atopic dermatitis is always worse before an examination. I think a good deal of the necessary psychotherapy can be done by the pediatrician, but if it is obvious that there are deep-seated and long-standing psychiatric difficulties, he will do better to send the patient to a psychiatrist, for techniques which we do not have are necessary in dealing with such patients.

ATOPIC ERYTHRODERMA

What I have called "atopic erythroderma" is atopic dermatitis, but it is a rather special form of atopic dermatitis, and warrants separate discussion. It may begin in a baby 3 or 4 months old, exactly as Leiner's disease does, or it may develop from a severe and generalized atopic dermatitis. The skin is bright red all over, usually with extensive scaling. Vesiculation is not prominent. A marked general glandular enlargement is one of the most characteristic features. These glands, particularly in the axillae, may be as large as walnuts, and often break down and suppurate. Furunculosis, otitis media, pyelitis, and pneumonia are common. One striking feature is a blueness and coldness of the hands and feet; I think that this and the general glandular enlargement are the most important diagnostic signs. There is always a high white count even when there is no fever or other gross signs of infection. Most of the increase in white count is made up of eosinophils, which may reach 50 per cent of the total white cells. There is usually a strong seborrheic element, and the appearance of the skin may sometimes be almost exactly that of Leiner's disease.

I look upon this syndrome as a combination of atopic and seborrheic dermatitis, and in the last few years have become convinced that overgrowth of staphylococci or streptococci on or in the skin and probable sensitization to them plays a dominant role in the clinical picture, and is the main reason why treatment is so unsatisfactory. The general glandular enlargement, the tendency of these glands to suppurate, the frequent occurrence of furunculosis, the high white count, and the frequent episodes of fever, all point to infection. Furthermore, it is not uncommon for the mother to develop small infected papules or pustules on her left forearm where the baby rubs his face when she is holding him, and she holds him a good part of the time because she has to rock him in order to keep him quiet. These babies not uncommonly die. A few years ago I went over the autopsy protocols of twenty-six of them who had died in the Infants' Hospital. Nearly all died from infection, plus poor nutrition—septicemia, pneumonia, pyelitis, or otitis media. The most common organism recovered was the *Staph. aureus*.

Treatment.—The two most important things in treatment are to eradicate infection in so far as it can be done, and to keep the baby in as good general condition as possible by giving him plenty to eat. He is in for a long sickness, no matter what is done, and adequate nutrition is of great importance to enable him to weather it. These babies are the most highly allergic people there are; their degree of sensitivity to egg white is terrific, and every one of them that I have been able to follow has later developed asthma or hay fever.

In years gone by I did fairly complete indirect testing on twenty-three of these babies. There were reagins to egg white in twenty-three, to wheat in nineteen, and to milk in ten, and occasionally reagins to other foods and to dust and feathers.

Removal of foods or environmental allergens does little or no good—these patients do no better in a hospital than at home. I am sure, how-

Fig. 25.—Atopic erythroderma without scaling. Local treatment: petroleum jelly, washing with pHisoHex.

Fig. 26.—Atopic erythroderma with slight scaling. Note enlarged glands in groin and poor nutrition.

Fig. 27.—Severe atopic dermatitis, changing later to atopic erythroderma. Before extensive scaling. Note normal skin in diaper area. (From Brennemann's Practice of Pediatrics.)

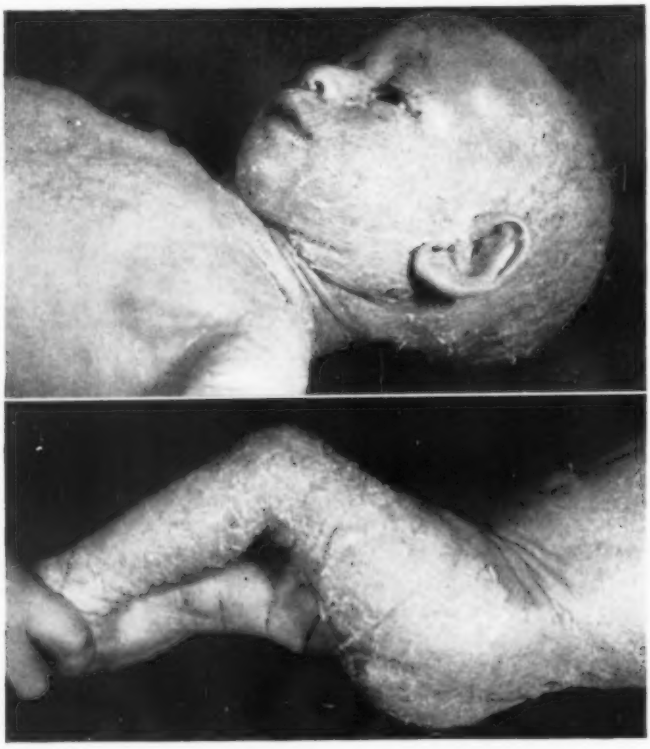

Fig. 28.—Atopic erythroderma. Same patient as in Fig. 27, three weeks later. Local treatment: petroleum jelly with 5 grains of salicylic acid to the ounce. (From Brennemann's Practice of Pediatrics.)

ever, that if they ate egg white they would have violent symptoms, and it is best to give them no wheat. A milk-free food does them no good; indeed it may do harm, for diarrhea is to be avoided at all costs. For over ten years I have done no skin testing either direct or indirect on these babies and am able to treat them better now than I did previously because after a good deal of useless dietetic treatment I have come to realize that food sensitivity is not the main cause of their trouble. For some years I have given them the diet mentioned before, consisting of milk, rice or oat, lamb, banana, squash, string beans, carrots, apple sauce, and Vi-Penta drops. I think they do better on a formula with half the cream removed, and with 3 or 4 added tablespoonfuls of Casec to the quart, as discussed under Seborrheic Dermatitis. I have also thought that large doses of ascorbic acid (100 mg. a day) possibly did some good.

Reduction of the bacterial flora of the skin and the prevention of any localized pyogenic infection are of prime importance, and it is best to give moderate doses of some antibiotic right along. This, however, does not produce any striking results.

Local treatment likewise is directed at antisepsis of the skin. For the arms and legs I use 2 per cent Vioform either in petrolatum or in zinc oxide paste, depending upon the condition of the skin, or a mild tar paste. For the face, the same, and for the trunk, a mild vanishing type cream or plain petrolatum. I like to have these babies washed twice a day with pHisoHex, which is a mild antiseptic and may do some good. An alternative to this method of treatment is to dip the baby twice a day in a bath of 1 to 10,000 potassium permanganate solution and let him stay there ten minutes. I have sometimes seen this do very well, particularly if there is a tendency to oozing.

I think these measures do some good, but progress is slow no matter what is done. These babies are never cured quickly. Toward the end of the second year they recover. One of the first signs of beginning recovery is that the feet are no longer blue and cold. Then the enlarged glands begin to go down, the erythroderma gradually lessens and white patches of skin begin to appear. It is my impression that they do not have atopic dermatitis later in childhood, but I have not enough accurate figures to be sure of this.

THE LOCAL TREATMENT OF ATOPIC DERMATITIS

Although sometimes much good is done by immunologic treatment, and it is certainly worth while because it attempts to get at the cause of the disease, too often it does not actually accomplish much. When all is said and done, skillful local treatment is more valuable than anything else. It is not necessary to have at your command a large number of preparations. The proper use of a few is more important, and after you have treated many of these patients you begin to see what really works and what does not. It is important to bear in mind that at any given time the eczema may be in different stages on different parts of the body, and one part may need an entirely different treatment from another part. In general, mild

preparations are used for acute eczema, strong preparations for chronic. My favorite lotions, ointments, and pastes are as follows:

I. *For Acute Oozing Eczema.*—

If there is much oozing, a clear watery lotion should be used—not a shake lotion or a salve. It is best used as

Fig. 29.—An oozy face which has been treated with a shake lotion. This should not be done. Proper treatment is with Burow's solution, camomile tea, or potassium permanganate. (From Brennemann's Practice of Pediatrics.)

a continuous wet dressing, or if this is not possible, it should be sopped on very frequently.

(1) Burow's solution 1 tablespoonful
Water 1 glassful

(2) Potassium permanganate 1 to 10,000
Potassium permanganate gr. vi
Water ʒ iv
1 teaspoonful to 1 quart of water gives about a 1 to 10,000 dilution of permanganate
or
A 5-grain tablet to a gallon of water gives about a 1 to 12,000 dilution

(3) Silver nitrate solution, 0.25%.
This is used for small areas only.

(4) Camomile tea. This is prepared by steeping 4 teaspoonfuls of camomile flowers in a quart of boiling water for 20 minutes. Then strain.

II. *Shake Lotions.*—

There are innumerable shake lotions. I use the three below more than any others.

(1) Burow's solution ʒ iv
Zinc oxide
Talc āā ʒ i
Glycerin ʒ vi
Lime water ℥ iv

This is somewhat astringent, soothing, and does not contain much solid material. It is useful for slightly moist areas where two skin surfaces come together as in the front of the neck, axillae, and groins.

(2) Calamine lotion (U.S.P.) (without carbolic)

(3) Liquor carbonis detergens (N.F.)
Glycerin āā ʒ iii
Calamine lotion (U.S.P.) ad ℥ vi

These lotions are useful for rather mild, extensive, maculopapular eruptions on the trunk. If tar is tolerated No. 3 is better than No. 2. This lotion is painted on with a small ordinary paintbrush. If it is too drying it can be alternated with one of the mild "creams" listed below.

III. *Ointments and Pastes.*—

(1) Acid Mantle Creme*
(2) Neobase†
(3) Hydrophilic ointment (U.S.P.)
(4) Petroleum jelly
(5) Toilet lanolin‡

None of these has any great therapeutic action. They are used where the skin is dry, scaly, and where a mild soothing and lubricating action

*Dome Chemical Co.
†Burroughs Wellcome & Co.
‡E. R. Squibb Co.

Fig. 30.—Papular form of diffuse atopic dermatitis. Local treatment: calamine lotion with added liquor carbonis detergens. (From Brennemann's Practice of Pediatrics.)

Fig. 31.—Pruriginous form of atopic dermatitis. Local treatment: calamine lotion with liquor carbonis detergens. Lotions are better than salves for this type. (From Brennemann's Practice of Pediatrics.)

is desired. Many children over the age of ten years with long-standing atopic dermatitis have very definite ideas about what they like to use, and what their skin will tolerate. Most of them find that some mild soothing application, such as one of those above, give them as much relief as anything. The first three are water-soluble, the last two are not. Lanolin should not be used if there is sensitivity to wool. There is a great difference in what skins with long-standing atopic dermatitis can tolerate. For some, a greasy preparation such as Lanolin or petroleum jelly may be irritating; for others these work better than the water-soluble ointments.

Fig. 32.—Note thickening of palm and many fine crisscross lines. This appearance is seen in atopic dermatitis which has lasted a long time. It will continue to last a long time. Such an appearance of the palms is of rather bad prognostic import. (From Brennemann's Practice of Pediatrics.)

(6) Salicylic acid gr. xx
Petroleum jelly ʒ ii

This is sometimes useful after Swartz's ointment has been used and there are many small adherent shrivelled scales left on the skin. The use of this ointment softens them, and they can then be detached by washing with pHisoderm. Some older children with atopic dermatitis have very "dead," dry, thick, scaly skins all over which closely approach ichthyosis. Many have keratosis pilaris, which consists of many little horny plugs in the follicles, and is seen particularly on the outer surface of the upper arms and on the lower legs. Still others have "keratosis palmaris et plantaris," which is often hereditary, and which is shown by great keratotic thickening of the palms and soles. These three conditions cannot be entirely cured and they complicate the therapeutic problem, for such skins "eczematize" very easily from any sort of chronic external irritation, particularly in the winter. Ointment No. 6 is helpful; it should not be used over a large body surface at one time. In most

textbooks of dermatology large doses of vitamin A are recommended for ichthyotic skins and for keratosis pilaris. I have never seen it do any good.

The general character of the skin means a good deal so far as prognosis goes—if it is soft and smooth where there is no dermatitis, the prognosis is much better than if it is of the dead, dry, "asteotic" type. In some children with atopic dermatitis there is seen an exaggeration of the small crisscross lines, with or without thickening, that run across the palm of the hand. (See Fig. 32.) This indicates chronicity, and means, as a rule, that you are dealing with a stubborn and a difficult case.

(7) Swartz's ointment:

Salicylic acid gr. xxx
Mercurochrome crystals gr. xx
Aquae q.s.
Anhydrous lanolin
Petroleum jelly āā ʒ i

This was originated by Dr. Jacob Swartz of Boston over thirty years ago for the treatment of fungus infection, for which it is still good. I happened to use it once on a child who had no fungus infection, but subacute atopic dermatitis in front of the elbows and back of the knees. It worked so well that I have used it constantly ever since for such patients. I have never seen it irritate, and it almost always does good, for what reason I have no idea. It should be well rubbed into the skin and not simply spread on. If the dermatitis is thick and lichenified, a strong tar paste or ointment is better. (See Fig. 34.)

Fig. 33.—Subacute atopic dermatitis. Local treatment: Swartz's ointment. It almost always does remarkably well for this type. (From Brennemann's Practice of Pediatrics.)

(8) Vioform.

This is used as a 2 per cent ointment either in petroleum jelly or in hydrophilic ointment (U.S.P.), or as a paste in the same strength with paste zinc oxide (U.S.P.) as the base. It is a fairly good antiseptic and fungicidal agent, is usually well tolerated, and is not likely to sensitize. I have used it especially on the arms and legs of infants with atopic erythroderma, and sometimes for moniliasis of the abdomen and pubic

region. It is a good preparation, but I think its value has been somewhat exaggerated.

(9) Burow's solution 3 i ss
Hydrophilic ointment (U.S.P.) 3 vi
Paste zinc oxide (U.S.P.) ℥ i

This is a soothing, slightly drying, excellent preparation for acute dermatitis if it is not too wet.

(10) Lassar's paste (paste zinc oxide, U.S.P.)

This is an old and tried preparation of value. It has the virtue that the large amount of starch it contains enables it to absorb a little fluid, and it is so thick that it is protective. I use it for acute angry eruptions in order to get them calmed down a bit before using tar, and as a protection, applied thickly with a throat stick, for ammonia dermatitis.

(11) Crude coal tar.

This is the most valuable local remedy there is for most infants and children with atopic dermatitis. It can be used as it comes, undiluted, or in pastes or ointments made up in various bases, and of various strengths. My favorite preparations are as follows:

Crude Coal Tar Undiluted.—This is not used enough; people have forgotten about it, but it should be remembered that when coal tar was first introduced in Switzerland and in France about 1910, it was always used in this way. Its incorporation into ointments came later. Undiluted tar is used on small thickened areas anywhere on the body. It is painted on twice a day, and dusted over with talcum powder. It is very useful for the small cracks at the lobes of the ears, which are so common in atopic dermatitis.

Fig. 34.—Lichenified atopic dermatitis. Local treatment: a strong tar paste. (From Brennemann's Practice of Pediatrics.)

Tar Pastes and Ointments.—These, of various strengths and made with various vehicles, are unrivalled in the local treatment of eczema in infants and children. Sensitization is unusual. It is best not to use a tar preparation on a hairy area, or where there is any pustulation. Tar is photosensitive and should not be used on the face if there is to be exposure to bright sunlight or considerable irritation may result. The mother should be told of this. The more chronic and thickened the eczema is, the stronger the tar preparation should be. I have never seen any bad results from absorption, but have heard of them, and it should be remembered that tar is a complex substance, containing elements which are toxic if absorbed in large amounts. I have followed the rule not to use a tar preparation over a very large surface area, and have limited its use at one time to both legs, both arms and the face, or to the trunk.

My favorite preparations are the following:

(1) Crude coal tar ʒ i
Zinc oxide ʒ ii
Starch ʒ iv
Petroleum jelly ℥ ii

This is about 5 per cent strength, and is the original tar paste used by Dr. Charles J. White, as he introduced it at the Massachusetts General Hospital in 1912. It is black and dirty.

(2) Crude coal tar
Yellow wax āā ʒ i ss
Petroleum jelly
Lanolin āā ad ℥ ii

This is sometimes used on very thick chronic places (ankles or wrists), where there is no moisture, so a paste is not necessary, and it is desired to secure good penetration. It should not be simply spread on the skin, but well rubbed in. It is sometimes of advantage to add 10 grains of salicylic acid to the ounce. It is a strong ointment—I use it only occasionally.

(3) "Daxalan" ointment.*

This is similar in composition to No. 1, only a little weaker (3 per cent). It is a remarkably smooth and well-made preparation, and is prepared so much better than most druggists can make tar ointment that for a long time I have not used prescription No. 1, but have prescribed Daxalan instead. It is black and dirty.

(4) Kolpix "A" and Kolpix "D."

These ointments are also made by the Dome Chemical Company, and differ somewhat from the Daxalan ointment in that the tar has been treated in some way so that it is a medium gray color instead of black, and the base is not greasy but water-soluble. The strength of each is 2 per cent tar. Kolpix "A" is for acute, somewhat active eczema, Kolpix "D" for chronic eruptions. Neither is very dirty. These are fine preparations—I use them a great deal.

(5) Tarquinor.†

This is another fine tar preparation, also in a water-soluble base, and not very dirty. Its strength is 1 per cent, and it has added to it 0.20 per cent of Quinolor, an antiseptic.

*Dome Chemical Co.
†E. R. Squibb & Sons.

(6) Tar and bentonite pastes.

At the Children's Hospital about 1942, Mr. Donald Skauen, the hospital pharmacist at that time, in collaboration with friends at the Massachusetts College of Pharmacy, which is across the street, and with a few suggestions from me, developed a thick tar paste, which has proved very useful. It is made thick by bentonite, and smooth by adding a certain vanishing cream.

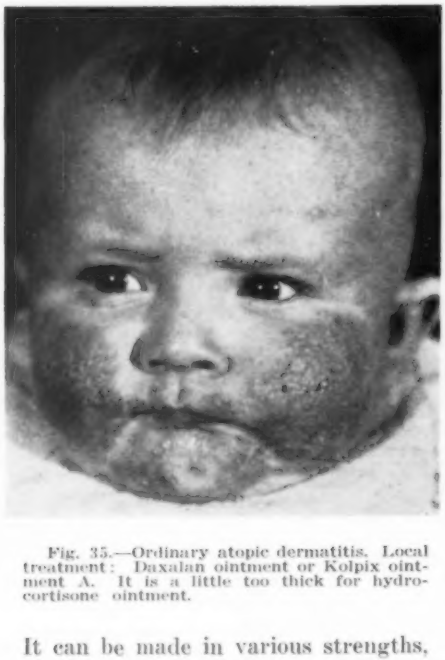

Fig. 35.—Ordinary atopic dermatitis. Local treatment: Daxalan ointment or Kolpix ointment A. It is a little too thick for hydrocortisone ointment.

It can be made in various strengths, and we have called it Tar and Bentonite Paste Nos. 1, 2, and 3. The No. 1 is very weak, the tar in it being derived from liquor carbonis detergens (N.F.); the No. 2 contains 2 per cent of coal tar, the No. 3 contains 5 per cent. These pastes are much thicker than the other tar pastes and stay on the skin better. I use the No. 1 a great deal when I want a protective, rather mild, clean preparation for use on places that are not much thickened. There is too little tar in this preparation for it to be of any value in chronic, thickened eczema. It is not dirty. I use the No. 2 and No. 3 occasionally, but am more likely to use Daxalan or Kolpix.

Fig. 36.—Deep papular form of atopic dermatitis. Local treatment: a 3 or 5 per cent tar paste. Hydrocortisone ointment does not do well for this type. (From New England J. Med., 1953.)

Tar and Bentonite Paste No. 1

Bentonite (U.S.P.)	7.50
Liquor carbonis detergens (N.F.)	15.0
Water	15.0
Special vanishing cream or "Neobase" or Hydrophilic ointment (U.S.P.)	7.50
Paste zinc oxide (U.S.P.)	45.0

For Paste No. 2, 2% of crude coal tar is used instead of the liquor carbonis detergens, and for Paste No. 3, 5% tar is used.

Special Vanishing Cream

Glyceryl monostearate	8.0
Spermaceti	7.0
Theobroma oil	5.0
Liquid petrolatum	8.0
Distilled water	67.0
Glycerin	5.0
Methyl parahydroxybenzoate	0.0625
Propyl parahydroxybenzoate	0.0375

(7) Hydrocortisone ointment (1%, 2.5%)

This is a valuable addition to the armamentarium, although I am not quite so enthusiastic about it as some have been. I have treated, to date, about 120 infants and children with it. It is of most use for inflamed, rather superficial, slightly scaly areas. It does not do as well when there is no active inflammation and a good deal of thickening—for such places a tar preparation is better. It should be applied twice a day, and gently rubbed in. The 1 per cent ointment in most cases works about as well as the 2.5 per cent and is much less expensive. Hydrocortisone ointment is not a cure—it simply reduces inflammation, but it does this with remarkable efficiency. If it has worked well, and after a few days there is no more eczema, it is discontinued until the eczema returns, and then resumed. If after a while the 1 per cent ointment does no good, then the 2.5 per cent can be tried. If this does no good, go back to the older methods of treatment. Hydrocortisone ointment is by no means a cure-all for eczema, and there is still plenty of room left for the older preparations that have been successful for years in local treatment.

Some hydrocortisone ointments contain an antibiotic; some do not. It is best, if there is any question that the lesion being treated is infectious eczematoid dermatitis, to use one of those that contains an antibiotic; several times I have seen such places develop gross infection after the use of the ointment without an antibiotic, and at least once when a hydrocortisone ointment containing neomycin was used. I think it best, if gross infection is present (pustulation), not to use hydrocortisone ointment either with or without added antibiotic, but to get rid of the infection by the means which have been previously described. So far as is known, no systemic effects are produced by hydrocortisone ointment.

Treatment of Eczema With Oral Cortisone

The oral use of cortisone has a place in the treatment of eczema in infants and children, but I do not think such an important place as it has in asthma. I have treated about sixty eczematous children with it, and have come to some fairly definite conclusions about it. In the first place, it is not a cure, and not infrequently when it is discontinued, the eczema returns and is worse and harder to get rid of than it was before. This is particularly true if it is used in patients where there is a bacterial element to the eczema, as there is in atopic erythroderma and infectious eczematoid dermatitis. I have come to the conclusion that it is best not to use it for atopic erythroderma, unless just for a few days to tide over some very difficult situation. The reason for this is that in atopic erythroderma it is necessary to use too large doses and to continue it over too long a period of time. When it is discontinued, the whole trouble

is likely to recur worse than it was before and may be harder to get rid of, so that the duration of the eczema may be actually increased rather than lessened by the use of cortisone. I expect that the reason probably is that bacterial growth is encouraged rather than inhibited. I do not advise its use for a baby who has blue feet with or without erythroderma, or for nummular eczema. I have found these things out by bitter experience. It ought not to be used if the eczema can be reasonably well controlled in other ways. It is, however, of very considerable value in certain situations. I have had several babies with oozy, crusted faces, which were kept inflamed by rubbing to such an extent that the usual methods of treatment were of no value. The oozing and itching were promptly controlled with cortisone, and did not recur to any great extent when it was discontinued. Another type of patient for whom cortisone is often satisfactory is the child of 3 to 5 years with a diffuse deep-seated papular eruption all over. Cortisone will usually relieve this quite promptly and give you and the patient a breathing spell, and a chance perhaps to find the cause of the eczema, which is often some specific food in this type of eruption.

Fig. 37.—Before cortisone treatment. (From New England J. Med., 1953.)

Fig. 38.—After one week of cortisone treatment. (From New England J. Med., 1953.)

Another indication is in the older child whose eczema is just so terrible that nothing can be done with it. He has been to a number of different doctors, has been dieted, innumerable salves have been used, and still he is no better. He is awake most of the night, and so is his mother, so that a very bad situation has developed in

the family. They all are worn out, and irritable, and immediate relief of the situation is necessary. Still another situation where cortisone is of value is in severe atopic dermatitis due to pollen, as in pollen eczema it is not necessary to continue the cortisone very long.

I have made it a rule never to give more than 75 mg. of cortisone a day to an infant or young child with eczema; if they need more than this I do not want to treat them with cortisone. I usually start with this amount, and in a few days when the eczema is controlled, as it usually is, drop the dose to 67.5 mg. a day for a few days, then to 50 mg., and finally to 37.5 mg. There are few patients with eczema who get any benefit from a dose smaller than this. To the babies under 2 years I have given 5 grains of potassium chloride daily; for older children I have simply prohibited the use of added salt to the food after it has been brought to the table. I have usually had the mother call me by telephone three days after the cortisone was started, have seen the child a week after it was started, and from then on every two to four weeks according to circumstances. I do not try to get rid of the eczema entirely, but simply to keep it under reasonably good control, as this can usually be done with moderate doses. It seems to me that the use of large doses is not justified in such a disease as eczema, because the trouble that may arise from large doses may be worse than the original disease.

There is a rumor around that cortisone stops the growth of children. As I write (April, 1955), nothing to my knowledge has as yet been published about this. I have kept growth records in the last three years of forty-three allergic children who have been treated with cortisone for periods varying from three months to three years. There were 417 months of cortisone treatment. During this time these children gained 80.75 inches in height. Forty-three normal children of the same age, height, sex, and

Fig. 39.—Cushing's syndrome, produced by only 50 mg. of cortisone a day. This is very unusual. (From New England J. Med., 1953.)

percentile height group would have gained in the same time 86.25 inches. This is a difference of only about 7 per cent. These figures have been examined by two expert statisticians and they reached the conclusion that from them it could not be concluded that the use of cortisone retarded growth, nor could it be concluded that it did not retard growth.

It is clear, however, that if growth is retarded it is not retarded enough

to amount to anything, which after all is what we want to know, and no child who needs cortisone should have it withheld for fear that it will dwarf him—it will not.

Epilogue

"It happens quite naturally that men who believe too firmly in their theories do not believe enough in the theories of others. We must disregard our own opinion quite as much as the opinion of others, when faced with the decisions of experience. When two physiologists or doctors quarrel, each to maintain his own ideas or theories, in the midst of their contradictory arguments, only one thing is absolutely certain; that both theories are insufficient, and neither of them corresponds to the truth. . . . We really know very little, and we are all fallible when facing the immense difficulties presented by investigation of natural phenomena."

—Claude Bernard: Introduction to Experimental Medicine.

Clinical Conference

INTER-UNIVERSITY ROUND TABLE CONFERENCE* BY THE MEDICAL FACULTIES OF THE UNIVERSITY OF PENNSYLVANIA AND JOHNS HOPKINS UNIVERSITY

Psychological Aspects of the Sexual Orientation of the Child With Particular Reference to the Problem of Intersexuality

DR. PAUL GYÖRGY.—Ladies and gentlemen, the Inter-University Seminar came about as follows. In July we had a little 9-year-old girl, or boy, in our ward, and I discussed the interesting findings later at a visit with Dr. Fremont-Smith in New York. He called my attention to the fact that intensive studies on problems on intersexuality have been carried out for the last few years at Hopkins, joint studies by the group headed by Dr. Wilkins from the pediatric point of view, and Dr. Whitehorn and his group, chiefly Dr. Hampson and Dr. Money from the psychologic-psychiatric aspect.

He recommended that perhaps it would be a good idea to send our patient over to Hopkins for study and when both of our groups have completed our studies we should get together in a kind of inter-university seminar, which we have today.

I would like to ask Dr. Stokes to make a few remarks as the chairman of the pediatric department.

DR. JOSEPH STOKES, JR.—We are delighted to have this Inter-University Seminar. I think probably the biggest problem in pediatrics as we look at the whole field is that of the psychologic development of children. In recent years, the groups that have been giving us the most information in relation to the mechanisms involved in pediatric difficulties, so far as psychologic development is concerned, are the pediatric endocrinologist, the neurophysiologist, and the psychologist, as well as the psychiatrist.

We do not know all the mechanisms involved in these intersex cases; certainly the pediatric endocrinologists have helped us tremendously. As we think of pediatric endocrinology it has seemed in the past almost like an occult science.

We are delighted to have Dr. Wilkins and his group here this morning. We are particularly thankful to Dr. Frank Fremont-Smith of the Macy Foundation for his help.

DR. GYÖRGY.—Dr. Rashkind.

DR. WM. RASHKIND.—The case for presentation this morning is that of a 10-year-old white child. This child was first admitted to the University of Pennsylvania Hospital on July 2, 1953, with the chief complaint, in her mother's terms, of a "growth on her privates." This growth was described and obvious at the birth of the child and, according to the mother, has enlarged uniformly and proportional

*Sponsored by the Josiah Macy Jr. Foundation, New York.

to the child's growth and development. At the time of delivery, the attending physician could not tell the mother just exactly what the sex of the child was. The mother, being strongly desirous of having a girl, decided to rear the child as a girl. She named the child Geraldine in her own admission for the contingency that if ever the child should turn out to have been a boy rather than a girl, the name could easily be shortened to Gerald.

Her past history revealed that the child was a 5-pound, full-term, spontaneous, home delivery. She was able to sit up at 6 to 7 months, but she walked only at the age of 3 years. She had measles and chicken pox at age 3, scarlet fever at age 5 and a tonsillectomy at age 7. The only additional significant fact in her past history is that the child reportedly had frequent recurrent attacks of croup and on this account was slow in starting school. Her formal education at the time she was admitted was a matter of a year or two of actual schooling. The social history will be presented in some detail and I will limit it largely to the family history. There are three siblings in the family, two boys and one girl. All three of them are perfectly normal in so far as their external genitals are concerned. The mother is of average build with nothing particularly remarkable about her except possible gross evidence of mental inadequacy. The father is reported to be a perfectly average man, rather on the large side. He is at present confined in a state penitentiary, having been convicted for an incestuous relationship with the female sibling.

On physical examination, this child impresses one as a child of short stature, of markedly impressive muscular development, attired in female garb, but strutting, walking more in the manner of a boy than of a girl. She is a friendly, cooperative, very poised child who does not seem to have any degree of anxiety over her condition. The significant positive physical findings are by and large limited to the external genitals and they will be described in some detail by Dr. Michie, and I will defer them at this time.

Laboratory studies that were performed on the child were by and large all within normal limits. The blood count revealed 81 per cent hemoglobin, 5,800 white cells with a normal differential. Blood chemistries showed a blood sugar of 106, blood urea nitrogen of 17; serum cholesterol, 161; basal metabolic rate of plus 18. An electrolyte survey showed also normal findings: sodium, 142 milliequivalents per liter; potassium, 5; chloride, 100; calcium, 11; phosphorus, 6. Alkaline phosphatase was reported at two different times as 13 and 9.

(At this point the patient was brought into the room.)

Additional studies that were performed involved assay of twenty-four-hour collections of urine for hormone studies. Initial report of the 17-ketosteroids in the twenty-four-hour urine sample was reported to be 8. A subsequent check showed this to be only 2. The gonadotropins were less than 8 mouse units and the estrogens were less than 2 mouse units. Consultation with the ophthalmologist showed perfectly normal visual fields and normal vision. The radiologic studies were all within normal limits.

Bone age was normal. Intravenous urogram was normal. Skull film was normal.

This presents the essential history and physical findings other than what will be described by Dr. Michie.

DR. ALEX MICHIE.—When I first saw this patient, my impression was that:

1. The left gonad, measuring approximately 0.5 cm. in diameter, was in the left inguinal canal near the external ring.

2. The right gonad, measuring approximately 0.5 cm. in diameter, was in the right inguinal canal near the internal ring.

3. The 4 cm. long phallus was a solid shaft with a well-developed glans which was covered with a clitoris-like foreskin.

4. The urethra opened in the middle of the perineum and was surrounded on either side by loose slightly wrinkled skin, the bifid empty scrotum. No labia minora were seen.

In conclusion, I thought that the patient had a marked hypospadias with bilaterally undescended testicles. Dr. György and I agreed that this patient had more masculine than feminine traits; hence, if she could be psychologically prepared for a change of sex, the hypospadias should be repaired and the testicles brought into the scrotum.

MRS. P. BROWN.—While Geraldine was a patient here on the ward, her mother was referred to Social Service for a discussion of the mother's feelings about this child and what the mother felt would be the outcome of a possible change of the child's sex. The mother kept three appointments with the social worker and was not able to come after that due to the distance involved.

She told us the family that she had was precisely what she had always wanted—two girls and two boys. Prior to the father's imprisonment the mother said that he was always abusive and alcoholic. She does not plan to reunite with him after he is discharged from prison.

The mother is a very pleasant young woman who enjoys her children thoroughly and enjoys playing games with them, particularly with Geraldine. She plays house with her and plays ball with the boys. There is a 13-year-old sister who at present is in the seventh grade, a brother of 12 who is described as being a wild child and the mother considered institutionalizing him. Since that time she has sent him upstate to live with an aunt and uncle and he is no longer in the home. He was in the second grade.

Geraldine started school when she was 8 and at present is in a special class in the public school. She has repeated the first grade three times. A brother who is 7 is also in the second grade.

The family live in a three-room apartment which is sparsely furnished, and their income is from the Department of Public Welfare.

The mother told us that when the patient was delivered at their home, the doctor told her that her child was a freak. She was very upset by this word, but she felt that because of this she would always want to protect Geraldine and to keep her from having other children being mean to her. An operation was suggested early in Geraldine's life, but this was deferred because the patient was so frail.

Later, when the mother suggested again that the child be operated upon, the father protested and would not permit her to go ahead with this.

The mother felt that Geraldine was different, but she felt that she was definitely a girl. She raised her as a girl and she feels that she is a very feminine child and has very feminine features. She said that the patient was not interested in playing ball, but her major interest was in playing house, playing with dolls, and in dressing up in feminine clothing.

She pointed out that the patient liked her hair to be long and in curls, and that she preferred ruffled dresses.

In discussing the question of Geraldine's possible change in her sex, the mother did not feel it would be possible to have the patient become a boy because she believes that Geraldine does everything as a girl does and could not learn to do otherwise. She said it would be better, in her opinion, to have a girl with a deep voice than have a very feminine boy.

DR. WISHNER.—Geraldine has been examined by means of psychologic tests twice—once on July 15 and once yesterday, January 20. The findings are as follows: On the Stanford-Binet Form L, with respect to intelligence, she is in the feeble-minded range. On July 15 she showed an I.Q. of 53, with very concrete thinking, very hard trying, good relationship to the examiner, but, nevertheless, feeble-minded; and we felt that this was an important consideration in evaluating the child's judgment and statements.

She shows little intellectual understanding of social situations despite her ability to relate to people, and at that time, on July 15, showed a good deal of anxiety about her present situation.

With respect to psychosexual identification, my conclusion at that time was that it was grossly ambivalent, that if either side was dominant it was the masculine side. The evidence for that was in the projective techniques, her tendency to identify ambiguous human figures as males or as both males and females.

In summary, then, the psychologic test data did not, in the beginning, point to any clear-cut subjective identification on the part of this child with masculinity or femininity; masculine components seemed slightly stronger. It was felt that the finding of feeble-mindedness should be a central consideration both in evaluating this child's statements and judgments and in determining her future sex. Postoperative examination possibly indicates some feelings of loss of maleness with concomitant anxiety; however, such an interpretation of this child's stories must be evaluated with considerable caution.

DR. M. DRATMAN.—This is a 10½-year-old girl who at times acts like a tomboy on the ward. She is mentally retarded and has a dull low-pitched voice sometimes heard in these children. This little girl spontaneously chose dolls and the doll house, fed the doll, put the doll to bed, and so on.

And why? Our interview with this child made it clear to us that her orientation and psychosexual development was feminine. The mother's history made it clear to us that she always desired Geraldine to be a girl and had treated her as such, even though she knew of the anatomic

difficulties from birth on. Her father considered her to be a girl, albeit he called her a "ruptured" girl. Her siblings and her friends considered her to be a girl.

It seemed to us that only psychologic disaster could follow if this child's gonadal tissues were allowed to determine her eventual sex. At 10½ the die is so firmly cast that it would be like saying to any one of us "give up your heterosexual orientation and become a homosexual." A totally impossible task.

Therefore, our conclusions, taking into consideration our psychologic report, were essentially: this child is psychologically a female and should so remain.

Dr. György.—At this stage this patient was sent to Baltimore. In short summary, there was a discrepancy between the pediatric and urological staffs, and the functional clinic. As you have heard, the psychologist has been on the fence, because he, at that stage, thought the patient had more masculine interests.

I myself have been greatly impressed by the behavior of the child, not from the psychologic or psychiatric point of view, but just the ordinary movements of the child—the gait, how she ran around the ward as a boy (not a tomboy, but as a boy). For I consider this child a boy, with a veneer of ten years of female surroundings and female education. However, I equally accepted the verdict of the psychiatrist that it would be a disaster probably to change the sex of the child. However, I repeat, I felt that the real sex of the child has been male.

Dr. Lawson Wilkins.—I think that I should say something first in regard to the anatomical and physical diagnosis of this case, then I would like to have Dr. Money and Dr. Hampson discuss the psychologic aspects. We hope that this case will be used simply as an example of how to study hermaphroditism from the anatomic, gonadal, endocrinologic, and psychologic aspects and then how to decide what ought to be done. And although the decision concerning the treatment and management of the patient depends partly on anatomic and endocrinologic findings, it rests to a greater extent upon certain psychologic considerations.

In the case of Geraldine, we have a child with a large hypospadic phallus resembling the male type. On external examination there was some question as to whether there was merely a hypospadic urethra or a urogenital sinus communicating with a vagina or vaginal pouch. Small nodules were felt in the inguinal canal and it had been assumed by some that perhaps these were male testes, but most of us were not sure whether they were gonads or lymph nodes. It was first necessary to obtain more information concerning the anatomic and endocrinologic status.

In so far as the external genitals were concerned, this child could have been a female pseudohermaphrodite with congenital virilizing hyperplasia of the adrenal or could have been some type of intersex without an adrenal disorder who had either testes, mixtures of testes and ovaries, or ovaries alone. The differential diagnosis between female pseudohermaphroditism with adrenal hyperplasia and intersexuality of other

origin is always the first thing which must be established. Actually, the distinction should be and can be made in the first month or two of life because in virilizing adrenal hyperplasia it is important to begin cortisone therapy early to prevent masculinization and permit a female with this condition to develop along entirely feminine lines.

As shown in Table I, the differentiation between female pseudohermaphroditism due to adrenal hyperplasia and intersexuality not due to the adrenal cannot be based on the anatomic structure of the external genitals or of the genital tract. The females with adrenal hyperplasia are born with a large clitoris resembling a hypospadic penis. They usually have a urogenital sinus communicating anteriorly with the urethra and posteriorly with a vagina, uterus, and tubes, but occasionally they have separate urethral and vaginal orifices. Now, exactly these same anatomic findings may be present in intersexes who may have testes or combinations of testes and ovaries (and occasionally ovaries alone). Of course, extensive surgical exploration of both the pelvis and the adrenal regions with biopsies of both gonads and adrenals might yield the diagnosis, but this is not necessary. The diagnosis can be made with certainty on the basis of whether or not there is evidence of secretion of excessive androgen. Within the first few weeks or months of life one can make the diagnosis on the level of urinary 17-ketosteroids. Normally at this age the output is less than 0.5 mg. per day. In patients with congenital adrenal hyperplasia the output is usually 2 to 3 mg. per day or greater. As the child grows older, the 17-ketosteroids rise still higher and the effects of excessive androgen secretion are manifested by excessive growth and accelerated epiphyseal development. By the age of 1½ to 3 years, pubic hair usually appears. Without cortisone treatment, viriliza-

TABLE I. DIFFERENTIAL DIAGNOSIS OF ADRENAL HYPERPLASIA AND TUMOR IN FEMALES

		EVIDENCE OF EXCESSIVE ANDROGEN			
	ABNORMALITY OF EMBRYONIC SEX DIFFERENTIATION	INCREASED 17-KS	EXCESSIVE GROWTH AND OSSEOUS DEVELOPMENT	PRECOCIOUS SEX HAIR	RESPONSE OF 17-KS TO CORTISONE
Congenital adrenal hyperplasia (female pseudohermaphroditism)	Hypertrophied clitoris persistent urogenital sinus (usually)	+	+	+	+
Genetic intersexes with testes, ovotestes, or ovaries	Various abnormalities (including the above)	0	0	0	0
Postnatal adrenal tumor	Normal at birth Later virilization and hirsutism	+	+	+	+
Adrenal hyperplasia	Same	+	+	+	+
Constitutional hirsutism or "premature pubarche"	None	0 or ±	0	±	0

tion and hirsutism invariably continue to increase throughout life. Unlike patients with adrenal hyperplasia, intersexes of other types do not show evidences of any sex hormones secretion in their prepuberal years. The 17-ketosteroids are not increased. Growth and osseous development are not accelerated. Sexual hair does not appear early. At the normal age of puberty secondary sexual changes may develop along either the male or female pattern irrespective of the nature of the gonad.

hyperplasia. Cortisone therapy would have no effect.

Table II shows a classification of types of sexual dimorphism. There has been a great deal of confusion in the literature in regard to terminology. Female pseudohermaphroditism due to virilizing adrenal hyperplasia can be regarded as a "sex reversal" occurring in embryonic life. I cannot go into the various theories in regard to the causes of "intersexuality." Among these cases there are "true hermaphrodites," "male pseudohermaphrodites," and occa-

TABLE II. CLASSIFICATION AND DIFFERENTIATION OF TYPES OF HERMAPHRODITES

TYPE	TYPE OF GONAD	EMBRYONIC ABNORMALITY OF SEX DIFFERENTIATION	EVIDENCE OF EXCESSIVE SECRETION OF ANDROGEN			SECONDARY SEX DEVELOPMENT
			17-KS	EXCESSIVE GROWTH AND OSSEOUS DEVELOPMENT	PRECOCIOUS SEXUAL HAIR	
A. *Intersexes*						
1. True hermaphrodites	One testis and one ovary, ovotestes or combinations of ovotestes	Various abnormalities of genital sinus and/or external genitals	Normal	0	0	Masculine or feminine
2. Male pseudohermaphrodites	Testes	Various abnormalities of genital ducts, urogenital sinus and/or external genitals	Normal	0	0	Masculine or feminine
3. Female pseudohermaphrodites (rare)	Ovaries	Female ducts Urogenital sinus Large phallus	Normal	0	0	Feminine
B. *Congenital adrenal hyperplasia*						
Female pseudohermaphrodites	Ovaries	Female ducts Urogenital sinus Large phallus	++	++	++	Masculine

In the case of Geraldine, it is obvious that virilizing adrenal hyperplasia can be excluded. She is 10 years of age. Her height is about that of a 7½-year-old girl. Her bone age is 8 or 9 years. She has no pubic hair. Her 17-ketosteroids were 1.3 mg. per day in our laboratory. She is, therefore, an intersex rather than a female with congenital adrenal

sionally "female pseudohermaphrodites." We prefer to apply the term "true hermaphrodite" to individuals with mixed gonads irrespective of whether there is an ovary on one side and a testis on the other, or whether there are various combinations of ovaries and testes or ovotestes.

We could be sure that Geraldine was an intersex of some sort. The

chances were very great that she was a male pseudohermaphrodite with testes alone or that she had some combination of testes and ovaries, because intersexes with ovaries only are exceedingly rare. (I have seen only one case.) The question of the type of gonad could be settled only by surgical exploration with biopsy and histologic examination of whatever gonads would be found. We felt that this should be done as a matter of scientific interest and that both surgical laparotomy and exploration with a urethro- or vaginoscope would be done to determine not only the type of gonads but also the exact nature of the genital tract in order to determine what female components were present and whether the anatomic structure could be best adapted for male or female functioning.

All that I have said to this point is concerned with our diagnosis of the anatomic and endocrine aspects of this case. Our decision in regard to what should be done in a problem of this sort depends upon a number of other important considerations, largely of a psychologic nature. We shall try to point out that we do not think that the decision in regard to the sex in which an intersex is to be raised should be based primarily on the gonadal sex or on the chromosomal sex.

We asked Dr. Howard Jones, our gynecologist, to examine her. Dr. Jones is present, but if he does not mind I will tell you his findings to save time. The child was taken up to the operating room and under anesthesia it became apparent that there was a vagina that did open externally and Dr. Jones passed a vaginoscope or small speculum up this tube without difficulty and visualized the cervix. Then he passed a small catheter through the cervix and measured the length of the uterine canal, which was 3 or 4 cm. long. So he decided this child did have a vagina, a cervix, and a uterus.

DR. GYÖRGY.—Can we discuss the exploration and then we can come back to the basic problem. Dr. Michie.

DR. MICHIE.—At operation we found that a relatively large vagina (it measured approximately 2.5 cm. in diameter and 4 cm. in length) terminated in a transverse, firm, fibrouslike cord measuring about 0.5 cm. in diameter and 2.5 cm. in length. Attached to each end of this cord was a normal-appearing Fallopian tube. Imbedded in the broad ligament and located slightly caudal and slightly mesial to the fimbriated end of each tube was a yellow gray ellipsoid mass measuring 1 cm. long and 0.5 cm. in diameter. In addition a vas deferens seemed to arise from the left within the broad ligament and left it at the junction of the middle and left lateral third. At that point the vas deferens was joined by a vascular pedicle. The 1 cm. free portion of the vas deferens ended in an epididymis which was attached to a testicle measuring 1.5 cm. in diameter. The testicle, epididymis, glans of the clitoris, and appendix were excised. The "ovaries" were biopsied. Following extensive exploration of the right internal ring we could not find another vas deferens or vascular pedicle. Following this the patient made an uneventful recovery.

In order to avoid our original error of calling a urogenital sinus a urethra, we now outlined the wall of the urogenital sinus with x-ray films. An

intravenous urogram was done. When the bladder is filled with urine containing the dye, we fill the rectum with air and the urogenital sinus with a radiopaque oil (Iodochlorol). By rotating the pelvis and by taking x-ray pictures in the various positions, it is possible to outline the walls of all three structures.

DR. GYÖRGY.—Can we have the sections?

DR. WM. YAKOVAC.—This tissue is labeled left ovary and nowhere in the section were seen any elements suggestive of ovarian tissue. Instead we found adipose tissue, connective tissue strands, blood vessels, a few nerves, and the like. To save time I can say the specimen on the opposite side showed the same thing. This is labeled testicle and here we find seminiferous tubules, or what we call in our lab a prepubertal testes.

DR. HOWARD JONES.—One is always worried when there is a unilateral gonad that one is overlooking the gonad on the opposite side, but in some instances, of course, there is but one.

In connection with the examination of these individuals there is a useful procedure when water will not stay in the vagina for the panendoscope. It was originated by a Philadelphian and graduate of the University of Pennsylvania, and is very useful. It involves turning the patient upside down and letting air fill the vagina, thus allowing an examination of the vagina with the endoscope that bears the name of the Philadelphian, Dr. Howard Kelly.

DR. WILKINS.—You have heard all the anatomic findings. I think we could sum up by saying that this girl had a testis, and a pretty good-sized phallus, which could have been made into a fairly normal phallus by a hypospadic operation, but that she also had a well-developed vagina which could be functional.

So if I had seen this child at birth or in the first few months or first year of life, the decision would perhaps have been difficult, in a way, because anatomically she could have been constructed either as a male or a female. But now still another problem entered into it. This child had been raised as a female up until now and had testes. Is one going to go on the type of gonad and say this is a male and must live as a male, or is one going to take the skin biopsies, such as are being done now by Barr in Western Ontario, and determine the chromosomal sex? Those skin biopsies would have come back a male chromosomal pattern, I am pretty sure. So this is a male.

What are we going to do about it and what are the questions which govern the decision? That is really the subject of this whole discussion today.

So we turned the case over to Dr. Money and Dr. Hampson who worked as a team and they examined and went over this child and discussed the problem with us and we abided by their decision as we do right along.

DR. J. MONEY.—Dr. Hampson and I counted up the number of cases that we have interviewed during the past two years and found that it is forty-four. We have no hesitation in saying that the general impression that comes unequivocally from this stack of evidence is that these people belong to the sex which was assigned

to them on their birth certificate, the sex for which their hair was cut and their clothing bought.

We have not come across anyone requesting a change of sex and a check of the literature, which has been quite comprehensive, indicates that these requests are very few and far between. But they do occur, so let me not sound too dogmatic. The general statement we can make is that a child's psychologic outlook, sexually speaking—his psychosexual orientation—is a product of or is determined by the kind of life experiences that this child encounters and the kind of life experiences that he transacts. That wording, you will notice, is careful not to talk about either heredity or environment, the one versus the other.

The kind of life experiences that a child encounters and transacts depends to a certain extent on the kind of body morphology that the child has, because the kind of sex that is written on the birth certificate after all does depend on an inspection of the genitals. In the case of some of these ambisexual children, it may be a superficial inspection, but inspection it is, nonetheless. And so the preliminary of any psychologic discussion, we feel, is a careful consideration of the morphology of the external genitals and the way they fit in the assigned sex.

We have seen some cases where the external genitals are completely unsuitable for the assigned sex, even though the gonads are the same sex as the assigned sex. These boys usually without any penis whatsoever have a very difficult psychologic adjustment to make, which is almost a self-evident statement.

Then the other preliminary to be considered is what we call the hormonal sex, that is, the kind of puberty that this individual will go through, whether it be a virilizing or a feminizing one. Dr. Wilkins makes, as you heard, a clear distinction between the female pseudohermaphrodites with the adrenogenital syndrome and other intersexes, and practically speaking this is a very important distinction because there is a possibility in cases of adrenogenital syndrome of predicting that their eventual pubertal development will be virilized and that it can be corrected with cortisone treatment.

But there are many cases for whom one must simply wait until the puberty period comes along. It may not be possible to predict before hand even after surgical exploration, because sometimes the gonads, although testicular in structure, are estrogenizing in their activity and effect.

This preliminary consideration of body morphology and pubertal development leads us on to a consideration of whether the individual grows up to have conviction or doubt about his or her sexual status. Obviously this will depend on and be quite closely related, in some instances, to the appearance of the external genitals. But there are other things, too.

Some children that we have studied have been brought up in secluded circumstances, with strict, rather moral environment, so that it is a long time before they discover the difference between boys and girls and quite late in their life before they are really fully aware of just how different they are. From the point of view of their sexual orientation this seems to be

helpful to them because it leaves them in no doubt for several of the formative years of their lives.

There are other children whose parents are perfectly straightforward and frank with them and talk about their medical condition with them and this also leaves them in no doubt, if it is properly done. Dr. Hampson is going to have some more to say about that.

By contrast you have the other child who is continually exposed to debate within the family, sometimes subtle debate and half secret, but within his awareness nonetheless and he is pretty much convinced that nobody is quite sure what sex he belongs to. The brothers and sisters tease him and the kids at school find out about his peculiarity and he gets teased there. Our experience has shown us that these children are in about the worst boat of all.

There is one exception. The ones that seem to be in the worst boat of all are those that have a change of sex imposed upon them without any consultation, and without any explanation. Dr. Hampson is going to talk about the pros and cons of a change of sex, so I will leave it at this point.

Now a word or two in conclusion about the general psychologic adjustment and psychologic stability of the individuals that we have seen. We found that 75 per cent of them have been, to all intents and purposes, well adjusted, and this includes all the cases where change of sex has been imposed, too. Those who have been subjected to change of sex or who have been exposed to debate and left in a state of ignorance and doubt about their condition, and a few others who developed some kind of psychologic instability for reasons that we cannot particularly track down, tend to have one particular kind of reaction which can best be described, I think, by saying that they have a rather circumscribed diffidence—circumscribed in that it pertains to the circumstances in their lives where their sexual status is liable to make a problem for them.

They are pretty careful not to get into too much social activity where dating and possible courtship advances would be made, and the emotion that is characteristic of these people, especially when they come up against a touchy situation, is the emotion of dread.

When you meet them for the first time the thing that is striking about them is that they seem to be painfully shy beyond all of the usual limits of excruciating shyness and it is difficult for them to open their mouths and talk about themselves, especially their sexual condition. With a little bit of help they usually overcome it when they find that this is a different situation and it is someone they can trust. They usually have not had anyone they could talk to and trust, as a matter of fact.

Along at the other extreme of the psychologic reaction, instead of this diffidence and dread, you find people with a reaction of grim determination that they are going to get through. I think probably the grim determination is the thing that occurs in those few who demand a change of sex. We have studied a man of 37 who determinedly changed from being a girl at 16. Some of those who are best adjusted to their assigned sex and most

stable psychologically also show a grim determination that they are going to cope with this problem of theirs.

Now for the second item on my agenda, our psychologic study of Geraldine. Rather than bore you, I shall read only our psychologic summary and appraisal of the child, and omit the first six pages of documentation:

"There was absolutely no question in the mind of either interviewer, even before cystoscopic examination revealed the presence of some müllerian organs, that this child should remain living as a girl. Psychologically she is entirely a girl, to which the following bears testimony. First, she displayed habitual mannerisms of demeanor and deportment—posturing, gesturing, position of limbs when sitting and talking, inflection and rhythm of speech—that were entirely feminine. Such mannerisms become so deeply ingrained after ten years that they are virtually ineradicable, especially when the person has a low IQ rating. Should the child live as a male, these mannerisms would create the impression of a very sissified male homosexual. It is pertinent to note that every physician who had dealings with her on the ward felt, upon unpremeditated first impression, that it was impossible to envisage the child as a boy.

"Second, the child demonstrated in her general play and social activities as well as in her verbal testimony that her spontaneous interests, recreations, and inclinations are girlish. In a manner quite typical for a girl of her age, she despises boys and boyish activities as being too rough and uncouth. She showed a girlish sense of modesty, so that one simply could not conceive of her being able to get used to the idea of using a boy's latrine.

"Third, despite the difficulty of obtaining extensive information about dreams, fantasies, and peripheral thoughts from a mentally defective child, the girl gave clear evidence in the TAT stories of a feminine orientation toward men. She unthinkingly regarded females as constantly in peril of being suddenly scared or assaulted by men. Though this orientation was doubtlessly connected with a drunken, bad-tempered, incestuous father, the significant point is that the child automatically placed herself in the female role while narrating the stories.

"Fourth, and most important, the child unhesitatingly regarded herself as a girl, and consistently chose to remain a girl and not to change and live as a boy." I will break my promise about documentary evidence here to say that in one oblique kind of inquiry that was conducted she was asked: "If some blind person came to you and said: 'Are you a boy or a girl?' What would you say?" Her reply was, "I'd know I was part boy. But I wouldn't tell him." She would tell him she was a girl, she added.

"All available evidence concerning change of sex in hermaphrodites strongly contraindicated change after the age of learning to talk. At a later period a change of sex is, with few exceptions, conducive to severe psychologic maladjustment and neuroticism. The exceptions are notable when the change is voluntarily requested and the patient is beyond middle teenage. Even so, in these people who change after the middle teenage, their reorientation is not

psychologically pervasive—the person acts his new role, maybe as convincingly as an actor who becomes the character he portrays, but is always capable of leaving the stage to become his authentic self. From the evidence so far accumulated, one must infer that most of a human being's sexual role is learned and in the early years becomes so deeply imbedded that a child cannot make a change of sex without paying too great price in psychologic disorder."

DR. WILKINS.—We have covered pretty well this point of whether one should change the sex of this patient who has been raised as a female to the age of 10. Well, the answer is no. Then what should we do with the gonads? Should we leave the testes alone or remove them? It seemed pretty obvious to us that we should not leave them if we are perfectly certain that we were going to keep this child as a girl. One cannot predict by any means whether the testes of intersexes at puberty will secrete predominantly androgenic or estrogenic hormones. Therefore, I think it would be disastrous to take a chance that, having made the decision that this child should remain a girl, she might a few years later begin to develop hair all over her chest and whiskers and various other masculine attributes. So our recommendation was that the testes be taken out and, as you have heard, that was done.

I am not certain of the legal status of the problem. So far as the ethical status is concerned, there is not any question in my mind. I think, however, the decision in regard to raising a person in a sex opposite from the gonads does depend upon what will be the outcome from the standpoint of psychosexual development. If we are going to raise an individual as a female in spite of male gonads and a male chromosomal pattern merely because there is no penis or anything that could be made into a penis, we must be certain to what extent the whole psyche and psychiatric pattern of the individual is dependent upon hormones, upon gonads, and upon chromosomes and to what extent they are dependent upon environment and the method of rearing.

DR. JOAN HAMPSON.—Dr. Wilkins has already discussed the criteria that he uses in the decision about what sex the neonatal infant should be raised in, if he is asked to make some such decision. I want to spend a little time in considering the child who is older than a neonate—perhaps a relatively young infant, but still not so young that he has not had a birth certificate filled out, not so young that the parents have not sent out announcements telling their friends "we have a boy" or "we have a girl."

I want to stress the point that Dr. Wilkins made that the external appearance of the genitals is an important factor in the older child, too. And not just the external appearance, but the mechanical potentialities for what the surgeons can do with what development there is—or is not!

Again, there is a sort of pre-selection in the cases that we get because it is, after all, the custom in medical circles of relying so much on the appearance of the external genitals in making their original decision "this is a boy." If there is something that looks like a good penis, the child is labeled a boy. Later there may be questions raised.

It is rare, on the other hand, for a child to be called a male if there is absolutely no vestige of a penis. So when we see the children, we are usually seeing children who have been assigned to a sex that bears some sort of relationship to the external genital appearance.

Dr. Wilkins also mentioned that in evaluating the various factors one should try to get the best possible estimate as to what the hormonal development at puberty or in later childhood is going to be in an individual. It was, of course, vastly more important before cortisone. When the female pseudohermaphrodite child faced a future of inevitable virilization, this, I think, was a very weighty matter.

If the female pseudohermaphrodite child had a well-developed phallus which could be constructed into something closely resembling an adequate penis, if there was no external vaginal opening, if you knew that the child was going to face a virilized future—big structure, big boned, bass voice, growing facial hair and sexual hair at an early age—it would be a logical conclusion, then, to raise that child as a male. However, since cortisone these issues do not come up often.

We do have some children upon whom the decision to raise them as a male was made before the days of cortisone. Should this decision now be reversed? We have had the case come up once or twice in children we have seen: the child who is gonadally female, but with adrenal hyperplasia, externally far more masculine looking than feminine and yet where with cortisone we could reverse all this and convert the child back to a biologically functioning female.

Well, the age of the child is the important thing, as our experience is beginning to show us. I might say now that we have seen only ten individuals in which there has been a change of sex. I think ten is probably a relatively large group for one group to study, but it does not give us any final authority and it would be unfortunate if I gave the idea that we have any final and dogmatic opinions about when sex should be changed and if sex should be changed.

The gonads themselves—these, of course, may or may not correspond with the potential hormonal development in the individual. I am sure that by now it is abundantly evident that we at Hopkins do not put much weight in gonads per se. As Dr. Wilkins has said, the gonad in the individual of sexual ambiguity is almost certainly infertile anyway. So far as libidinal direction is concerned, we now feel absolutely convinced that the hormone itself has nothing to do with the direction of the libidinal drive.

We have seen seven individuals who have been raised and are being raised in a sex which is contrary to their gonads. We have no evidence whatever that these individuals are facing problems of a libidinal direction different from what society expects in accordance with their assigned but not gonadal sex.

The important points that I want to stress, again, have to do with the sex of rearing and the duration of the rearing. We feel that age is important from two points of view—the child's and the parents'. We feel definitely that the sexual inclinations are so very much learned things and have so little relation to the hormones,

the gonads, and the chromosomes that the ultimate deciding factor in most cases is: How long has this individual been learning "boyness" or been learning "girlness"? What chance is there now that he can do an about-face and start learning to be the opposite sex?

The parents have to be considered, too. Parents learn about being the mothers and fathers of daughters; parents learn about being the mothers and fathers of sons. It takes a fairly flexible set of parents to be able to make a change after the first period of young childhood. It takes reasonably flexible parents to make a change at any time. We have never seen the results of any experiments, and I think it would be interesting to have one—make a change of sex and at the same time make a complete change of environment so that the effect of the parents would not be an influence on the child's chances of adjusting after a sex change.

I have been vague about sticking my neck out and saying exactly what age seems to be the critical age. I do not want to place too much reliance upon statistics, but we were looking at our figures the other day and found something that was quite interesting. In our ten individuals who have undergone at one time or another a change of sex, the change was made at various times ranging from 4 months to 16 years.

Of our total of forty-four cases of sexual ambiguity we have rated twelve of these individuals as having a wobbly sort of adjustment. In the others we have seen no evidence to have any debate about their emotional stability. So we have twelve out of our total forty-four. Five of those are of the ten who have made sex changes, so of our ten who have changed sex five have made only questionable adjustments to the change of sex. Their patterns of difficulty have been different, but there has been some reason to question the adequacy of their adjustment.

The other five (and we have followed them to a late enough age so that we have been able to make some kind of evaluation about their adjustment) have given us no evidence whatever that they were psychologically in any way suffering from the effects of their change of sex. There was no alternative but to evaluate them as thoroughly stable individuals.

The ages at which these people have changed sex emerged from our tabulation as follows: of the five who were rated as well adjusted, four were changed at less than 6 months of age. The other one was 2 years 3 months.

Of the five who are on our list of people with questionably satisfactory adjustments, all made the sex change at 15 months or older—two at 15 months, one at 4 years, one at 4½ years, and one at 16 years.

Again, I make no special statistical claim. This is a relatively small series, but I think it is big enough to make one think seriously about sex change after, shall we say, the age of learning to talk, after the age when a child has learned his name, after the age when a child has begun to have some idea of "I am a boy" or "I am a girl," after the age when the parent is thoroughly used to the idea that he has a son or daughter, after the siblings are used to having a little sister, after the neighborhood kids are used to seeing Jimmie running around in boy's clothes and with a boy's hair-

cut. Let us say that a year might be a fairly safe although arbitrary kind of border line to take.

I do not want to paint a too gloomy picture about sex changes, because the sex changes that we have seen in the young infants, in the infants who have changed at 6 months or so have been, so far as we have been able to evaluate them, entirely satisfactory.

One thing that I think needs the utmost emphasis, though, is that we have to consider in individuals who are old enough: What about the child's own preference? I think that we doctors get rather into a bad habit of making decisions about what is the best thing to do for people, though with their consent admittedly.

Surprisingly, some individuals are well able to talk about it in forthright terms, but we rely on evidence which is collected in more tangential and oblique fashions. We rely on the signs that the individuals give of their sexual preference and their sexual identification.

Geraldine today looked different than she did in Baltimore, with her hair drawn back into a pony tail. In Baltimore she insisted on having her hair up in curls which she tossed around with thoroughly feminine gestures. She glowed, just radiated, when I said, "My, what pretty hair you have."

I give this example as one of the signs that children display. Their mannerisms and inflections, their verbal patterns, the way they sit, their little almost unconscious gestures—these are other signs.

The daydreams and the fantasies as well as the reported night dreams we feel are very revealing. It is not always easy to find out about daydreams and fantasies in someone like Geraldine.

We have various oblique techniques that we use which are not formal projective tests, although we put some store on formal projective tests and we do use them. We like equally well the evidence that we get from what we call oblique tests—imagining, asking the child to imagine a situation.

"What would you do if . . .?"

"Tell me about ten years from now. Well, let me see, you'll be how old?"

Geraldine would be going on twenty-one, she said.

"And what would you be doing?"

"I'd be married."

Well, of course, one could say, "Yes, a boy might make this reply, too." And he might. But then one would have to inquire whether she was going to be the husband or the wife in the situation ten years from now. And by asking questions which scrupulously avoided suggesting to her whether she would be the husband or wife in this marriage ten years from now, it became perfectly evident, as was pointed out by Dr. Dratman's report, that Geraldine's thoughts of the future revolve around cooking and scrubbing and baking and fixing things and mending and the most domestic kind of activity.

Now, last and not least, the direct verbal statements of the individual, the conviction with which they can talk about being a girl: the foreignness that any kind of concept of being a boy means for someone who has been raised for ten years or so as a girl.

So I would simply say this, that though we are not committed to any absolutely definite point of view, we feel reasonably sure that if anyone

wanted to make a study of experimentally produced neurosis, it would be a surefire thing to take a child of more than 2, 3, or 4 years on up and subject them to a change of sex.

We feel that after the age of learning to talk it should be considered only with extremely serious scrutiny of the entire situation.

If there is some time, I think that it might be of interest, although it is not directly relevant, to say a few words about what we do in dealing with these individuals of ambiguous sexuality, and some of the ways, the practical ways, in which we can help the parents, and the sort of problems we find that parents face.

As will be now quite evident, we would plead for the earliest decision that is reasonably possible. And more than that even we would plead for decisiveness. One of the worst things that can possibly happen to the parents or to a child is to be sitting on the fence as to whether they have a son or a daughter. Our experience has shown that parents will not permit themselves to be left sitting on the fence. They make some kind of decision of their own.

So, again, the earliest possible decision—certainly under 6 months if it is at all possible, and, then, once the decision is made, let it be an irrevocable one. Let there be no halfway acceptance of this. Occasionally we have seen instances of sex change where, yes, the sex was changed and the hair was cut, the neighbors were told, the child was given another name, but, just in case, they were not going to change the birth certificate yet.

We have seen one child who has been known as Louis for years and years now, who has on his birth certificate (and he has to register for the draft soon so he is in a real dilemna), Mary Louise.

If this kind of thing goes on there is also, if not overt, then covert debate about it and this is a bad influence.

What practical measures can you remind the parents about? What will they ask you about and what should they be given some guidance on? Changing the birth certificate, insurance policies, and things like that.

The announcements: We had one extremely sensible, attractive young mother, wife of a West Pointer, who had a child who was changed in the very early weeks.

If a mother in her predicament can talk to someone who can discuss the matter calmly and without panic and who can give her some advice about sending out a whole new set of announcements, give her some advice about having some answers ready for the questions that are inevitably going to come, help her with an answer that is somewhere near to the truth and that is somewhere near satisfactory, too—that is quite necessary and important.

Another part of our job that we feel is important although mundane is finding out the parents' misconceptions about their child. Of all the parents we have spoken with almost invariably there has been some kind of fear that their child is really a "half-and-half," a "morphodite." Is this a Jorgensen sort of thing? Is this two-sexed, this child of mine?

We have found that parents can grasp the real situation in these cases of intersexuality much more accurately and much more satisfactorily for their own understanding if we talk about an unfinished child and

not about a half-and-half child. Because they are unfinished embryologically speaking.

We give the parents some simple embryology talks, with diagrams similar to the one that Dr. Wilkins used. We always come to the point in answering the parents' questions where we have to say we just do not know why, but before we reach that point we have cleared up a lot of the parents' misconceptions.

DR. GYÖRGY.—I think we should open now the discussion, but before we do it, I would like to make a few remarks. To summarize our point of view—I am speaking, I think, only for myself, but I am not afraid to stand up even to experts—I am a little taken aback by the revolutionary statement that gonads have nothing to do with sex. And in addition to it, although in all practical conclusions I fully agree with all that Dr. Wilkins from an anatomic point of view said and from a psychiatric point of view what Dr. Money and Dr. Hampson said—I fully agree. The only discrepancy is I still think that Geraldine was a boy and should be raised as a girl.

Now, I am a little disturbed, however, in my further remarks because the gonads, I think, have something to do with the sex and this child has now been emasculated because the gonads have been taken out. I will now ask the resident, Dr. Rashkind, what did she ask for as Christmas presents? Will you please repeat it?

DR. RASHKIND.—This child, when queried about what she wanted for Christmas, among other things asked for a holster (and these were rather prominent on her list) two guns, a guitar, and a tie.

I might also add that her favorite television program is Gene Autry with a close second being Hopalong Cassidy.

DR. HAMPSON.—I would like to make one point. I also asked her what she would like for Christmas. She said she would like two guns and a holster. She would also like a *cowgirl* suit to wear it on!

DR. WILKINS.—Dr. György brought up one exceedingly important point that I passed by entirely inadvertently, and that is that when the gonads are removed, of course something has to be done about secondary sexual development, otherwise the patient will remain sexually infantile. It is entirely in our power nowadays to bring about either male or female secondary sexual development with the proper hormones. Naturally, if we are going to raise this child as a girl, at the age that we select it would be important for her to develop breasts and other feminine sexual characteristics. She will be put on estrogenic therapy and eventually given cyclic therapy. If she has a uterus, as Dr. Howard Jones thinks, she will menstruate; if she does not have a uterus, she will not menstruate. So that question will be settled in a few years from now. If the patient were going to be raised as a male and had not had her gonads removed, we probably would not have removed them in this case. She would need testosterone at puberty.

DR. JOHN C. WHITEHORN.—It appeared to me that these cases that Dr. Wilkins has provide an unusual opportunity for evaluating some of the basic problems in modern psychiatry. We have ideas of personality development and growth in terms of cultural norms,

family patterns and attitudes, and systems of values. We have much evidence of the importance of growing up in an environment which is steady and consistent, and the disadvantages of growing up in an environment where there are conflicting attitudes.

We also have a good deal in our psychiatric literature with regard to instinctual impulses and sources of energy, the biochemical foundations of such instinctual forces; and it appeared that in Dr. Wilkins' material there would be a nice opportunity on psychiatric evaluations to find cases illustrative of endocrinological and biochemical ambiguities, even to make experimental changes in some of the biochemical and endocrinological forces—to have an opportunity there to evaluate the importance of biochemical instinctual factors and cultural environmental attitudinal factors.

Dr. K. Appel.—It is clear that in this patient we have bisexuality. We have the gun of Dr. György and the cowgirl dress of Dr. Hampson. Psychiatrists are familiar with this problem of bisexuality and here we have bisexuality embryologically and anatomically and psychologically and sociologically.

Our feeling is that libidinal direction does take place in spite of the gonads and personality development; behavior is determined, as we see here certainly in large part, by parental and social selection or guidance, by the experience the personality has lived through; so that we have to think both of biology and psychology and sociology when we are thinking of personality function.

Dr. Wilkins' selection on the basis of the pragmatic anatomic potentials of some sort of satisfactory future genital function, it seems to me, is an important thing. Also, the parental attitudes and wishes are important as we see here, because a determining factor in the orientation and social behavior of the child was the mother's influence.

If this boy were raised as a boy, he would be psychologically homosexual, I believe, and it seems to me that he would have more difficulties in that way than he would have in the decision that has been made.

Dr. Frank Fremont-Smith.—I would like to say that it has been a great pleasure to me to be here and to have a chance to listen to this splendid series of presentations. I would like to compliment everybody that participated all the way through. I think I rarely heard as good individual presentations and it certainly has made a fascinating whole; but also a whole in which there has not been complete agreement, thank goodness. Thank goodness to you, Paul.

I would like to say that I liked very much what Dr. Stokes said in the beginning in emphasizing cultural differences and the need of studying these needs in terms of the world concept.

I think also we have subcultural differences that need study and interpretation, and you find those between universities, and here in this seminar we are seeing two universities join hands on a common problem. Then we have within the university the subcultural divisions which are very, very difficult to get over, but here we see quite a lot of cross-departmental

cooperation; and to see this both within the university and in two universities is tremendously encouraging.

Being brought up in a biological rather than a cultural tradition of learning—of course, culture is Johnny-come-lately in our thinking—we assume that this boyness or girlness was biologically determined. But relativity, which has been brought vigorously into physics, is also a Johnny-come-lately in biology and medicine and we need to bring it in, and I would say that sex has several dimensions of relativity. Or the uncertainty could enter into some of those cases.

In those cases, then, it seems to be fairly clear from the evidence forcefully presented that the cultural determinants may and often do overbalance the genetic derminants. And, how, because I had to struggle with this problem many years ago of nature versus nurture and environment and organism and so forth, I finally came to the conclusion that you were dealing with something like a rectangle which had a length and a breadth. But it is obviously not a rectangle because you do not always have length and breadth. It is always the environment and the organism is nonexistent without it, and any manifestation of organism, any manifestation either in growth or behavior or development, is the result of the interaction of the organism as it was previously with the previous environment. Therefore, if you change the environment which is pertinent you are inevitably going to change the manifestation of this interaction.

Now, then, if I may revert once again to the depersonalized role of the Foundation, may I thank you very much indeed for letting me come and thank you for the exciting role. There seems to be a lot of unfinished questions and maybe there needs to be more seminars of this sort.

An Early Case of Sexual Orientation

Hartford January 1, 1770. We are credibly informed, that about 23 years ago a child was born in the Southern Part of Massachusetts-Bay, who bearing a similarity of both sexes, it was disputed what apparel it should be dressed in, but 'twas at least agreed to dress it in a Women's, and it was baptised by the name of Deborah; this person grew up, and till lately passed for a woman; but having for some time past lodged with one of that Sex, the latter found herself to be with child, and has swore the former to be the Father of it. . . . The consequence has been that they are married together, and the Father instead of his former name, was married by that of Deborah Francis Lewis.

FROM THE ESSEX GAZETTE, Jan. 30, 1770.

Pediatric Profiles

William McKim Marriott

(1885-1936)

NINETEEN years have elapsed since the death of McKim Marriott in 1936, a sufficient length of time to begin to consider his pediatric work and influence from an historical perspective. Shortly after his death an editorial in *The Journal of Pediatrics* told "of the meteoric career of one of the most brilliant minds that has flashed across the field of pediatrics in America . . . His influence will last for many years." Today, in retrospect, it is my opinion that in his career as a pediatrician, which lasted only a few years, Marriott was the most important individual influence that developed in American pediatrics in the first half of the present century. This feeling is based not simply on his important scientific contributions in the field of metabolism and the metabolic diseases of infancy and childhood, and to his influence on the many men and women who worked with and under him who have won distinction, but in no small part to his practical contributions toward the simplification of infant feeding. The principles of infant feeding which he taught and stressed have become in general the accepted methods used by the practitioner. Thus his influence extended far beyond scientific contributions to our knowledge and understanding of disease in childhood into the field of normal growth and development and practical "preventive pediatrics."

He was essentially a biochemist who embarked on a pediatric career by chance and as a matter of second

In writing this Profile of McKim Marriott, the addresses made by Drs. Philip A. Shaffer, Edwards Park, and Alexis Hartmann at a memorial service for Marriott held on January 3, 1937, at the Washington University Medical School, have been freely drawn upon. Dr. Shaffer, an intimate friend of Marriott for perhaps longer than anyone else, was asked to write the Profile but, unfortunately, was unable to undertake it, and thus the writing fell to the author, who was professor of clinical pediatrics at Washington University during the time Marriott headed the department as professor of pediatrics from 1917 to 1936. Dr. Shaffer would have done it much better. Additional suggestions and recollections by Drs. Park and Hartmann have been most helpful.

choice, entering the field through the side door, so to speak. His clinical training was scanty and he never became a "top clinician." The errors and mistakes that are found in his work can be traced back to this lack of clinical experience and training. While these have been emphasized by some, they are of minor importance when taken into consideration with his important contributions and his work as a whole. Up until Marriott's time individual progress and advancement in pediatrics was a laborious, slow procedure through the outpatient service and hospital wards until the top was reached by some. Marriott by-passed all this customary preparation for appointment to a chair in an important medical school, and was one of the first to set the pattern for the many physiologists and chemists who have been appointed to professorships of medicine, surgery, and pediatrics in recent years. The success that took place in this instance of the biochemist becoming an outstanding pediatric figure has had an important influence on the trend of medical education in the United States.

When Marriott was only 15 years old he was ready to enter college at the University of North Carolina at Chapel Hill. It was here, under Charles Baskerville, professor of chemistry, that he acquired his interest in and love for chemistry. In 1904, the year that Marriott received his B.S. degree from North Carolina, Baskerville was called to the chair of chemistry at the College of the City of New York, and took Marriott along as his assistant. Soon after reaching New York he met C. G. L. Wolf of the biochemical department of the Cornell Medical School. This led to Marriott's transferring to Cornell, where he spent the next six years learning the fundamentals of biochemistry under Wolf, assisting in the laboratory instruction, and, rather incidentally, attending enough classes to obtain his M.D. degree in 1910. His real interest was in biochemistry and he took no further hospital or clinical training.

In 1910 he went to St. Louis with Philip A. Shaffer and Walter Bloor to organize and develop the department of biochemistry in the newly reorganized Medical School of Washington University. In the following years, 1911 and 1912, the reorganized clinical departments took up their work in St. Louis. John Howland had been appointed professor of pediatrics and had selected me as his assistant and Kenneth Blackfan as resident. Howland's stay at Washington University was a brief one of six months. He was most unhappy, largely because of the distressing physical condition of the old Children's Hospital in the slums where he had to work until the new buildings were built on Kingshighway. The others in the clinical groups accepted the situation and made the best of it. In the spring of 1912 Howland decided to accept the offer of the chair at Hopkins from which von Pirquet had resigned. Blackfan went with him, while my decision was to stick by the St. Louis group.

During this and the following year, Marriott and I saw considerable of one another as we were about the same age and both part of the "junior echelon." We frequently lunched together at a saloon where we could get filling food and a glass of beer at an inexpensive price which was important to us both. He often talked of prob-

lems he wanted to work on which required work with patients. They were distinctly adult problems. Early in 1913 he went to Dr. George Dock, professor of medicine, and told him what he wanted to do, and asked Dock if he could make a place for him in the department of medicine. Fortunately for pediatrics, Dock told him, as Marriott related to me, "he did not want a chemist in his department." Marriott was quite depressed. One day the idea came to me that he join the pediatric department. After much discussion of the idea and of pediatric problems, and a complete understanding that we would have to learn pediatrics together, he decided to accept the idea. Early in May that year I told Dr. Howland of our plan. A few weeks later an illness sidetracked me for a "cure" that would require many months. Howland, when he heard of my illness, wrote that he was looking for a chemist for his department in Baltimore and asked if, under the circumstances, it would be all right for him to approach Marriott. It was not only "all right," but the letter solved a problem. It was in this way, by chance, and as a second choice, that Marriott went into pediatrics, and the association of Marriott and Howland came about.

This association in 1914 through 1917 developed a team whose work stands out as perhaps the most important influence on pediatrics that took place in the United States, and one that extended far beyond this country. Howland was an able teacher and clinician and his department at Hopkins soon became the mecca for young men who desired to become pediatricians. Important research began to appear. In discussing the association between the two, Edwards Park, who was at Hopkins during these years, wrote, "Howland remained the central figure in the department, but Marriott was the center of research thought." It was here at Hopkins, working with Howland, that two of Marriott's important contributions were made: the presence and importance of acidosis in alimentary intoxication, and the discovery of the low serum calcium concentration in tetany. No small part of his work was the development of technical methods of analysis and tests which made the clinical studies possible. It was Howland's intention and desire to keep Marriott wholly in the laboratory. To this Marriott would not assent and insisted on his having the opportunity for clinical training. Marriott told me once, in reminiscing over his Hopkins days, that he finally told Howland he would make a bargain with him. For everything that Howland taught him clinically, he (Marriott) would teach Howland something chemically. One gathers that it was a conflict of minds and purposes in which Howland had to acquiesce.

Around 1915-1916, through grants from the Rockefeller Foundation matched by local gifts, the decision was reached to put the departments of medicine, surgery, and pediatrics at Washington University on a "full-time" basis. By this time the new medical school buildings and affiliated hospitals were completed and in use. There were to be none but "full-time" men in the departments, who were to do no private work. The clinical groups did not look favorably on the "none but" part of this restriction, as they regarded it impractical and

not in the best interest of educating doctors. It was simply the time-worn futile argument of "research" vs. "training practitioners" as the primary purpose of the medical school. Both are important, and a medical school has a dual purpose which should not be the cause of conflict. The plan was put into effect in 1917 at Washington University. Some two years later, on returning from World War I, I found that the "none but" idea had already been discarded as impractical, if not impossible. Several names were suggested for the "full-time" chair of pediatrics, but for some time a decision could not be reached as the Rockefeller influence kept suggesting a certain man who was unacceptable to the University. Finally Dr. Shaffer, under whom Marriott had come to St. Louis in 1910, suggested his name. As no criticism of Marriott's research interests and accomplishments could be made, the chair was offered him in 1917. Despite contrary advice from Howland and his limited clinical experience, he accepted.

Soon, attracted by his budding fame and reputation, men from all over the country and abroad wanted to work in his St. Louis clinic and laboratories. Among names well known in American pediatrics who were associated with him on the pediatric staff, or worked under him for a greater or lesser period of time, were Samuel L. Clausen, P. C. Jeans, Jean V. Cooke, Alexis F. Hartmann, F. Scott Smyth, Milton J. Senn, Dan Darrow, Leonard Davidson, Hugh McCulloch, Russell Blattner, Rolf Struthers, Lester Evans, Harvey L. White, Martha Eliot, Clifford Bost, William Belford, Katharine Bain, and E. J. Christopherson.

Of those from abroad, Kirsten Utheim of Norway became the best known. An interesting item in the "Marriott memorabilia" is a memorial sent to the Medical School after his death signed by twenty names "representative of various universities in England and Scotland who had studied under him." It speaks of "the gratitude and gracious welcome to us on our arrival, and his warm friendship during our sojourn." It then goes on to say how they brought back "not only deep and lasting friendships, but a store of knowledge and the inspiration to acquire more knowledge as the result of our work under Dr. Marriott . . ." Some of this group were men on a yearly resident exchange basis with Leonard Findlay at what is now the Queen Elizabeth Hospital in London. The general conclusion of the men participating in this exchange was that the Americans learned much in the way of clinical observation and acuity in Findlay's clinic, and the group who came to Marriott's clinic learned much in the way of techniques and inspiration for research. Marriott never visited the European clinics but his work was well known abroad. Park relates how, in 1923, when Finklestein visited the United States, he told him that one of his ambitions was to have his photograph taken with Marriott.

Marriott's scientific contributions after coming to St. Louis centered chiefly around "alimentary intoxication" in infants, which was a logical development of his earlier Baltimore work on acidosis. Alimentary intoxication had been looked upon as a form of food poisoning. Its blame had been placed on various components in the diet, and a number of dietetic methods

had been introduced for its treatment, particularly in the European clinics. Marriott's contribution—perhaps his most important—was to show that alimentary intoxication was a group of symptoms brought about by a reduction in blood volume as a result of dehydration through water loss. This in turn led to the studies of the physiologic adjustments made by the body when dehydration occurs, which have been continued and elaborated by many workers since Marriott's time. In this work at St. Louis a number of his associates, as Utheim, took part, carrying out the details of various phases of the investigation under Marriott's guidance and inspiration. To this picture brought about by dehydration, he gave the name "anhydremia." It is excellently presented under this name in his Harvey lecture of 1923.

Perhaps his most controversial work was the relationship between diarrhea and infections of the middle ear. It was lack of good judgment of the medical ability of men, together with his own lack of clinical training upon which judgment is based, that led him to overstress and overemphasize the relationship between the two. I happened to drop into his office one day after there had been considerable criticism of this work and found him with an unhappy look on his face and two stacks of clinical records a foot and a half high on his desk. He put down the record he was studying and remarked he had been misled by the otologists. While such a relationship undoubtedly existed, particularly in those preantibiotic years, its importance was very much overemphasized by Marriott. Because of his prestige, undo importance was placed on the relationship and many antrotomies and mastoidectomies were being unnecessarily performed throughout the country. More difficult to understand is why Marriott, who had destroyed the concept that intestinal intoxication was a specific disease, should later in this work overemphasize a streptococcic infection in the mastoid.

One of Marriott's most important contributions to pediatrics was his work in the field of infant feeding as his teachings have had an important influence on the health of millions of infants in the United States which continues today. He became interested in the subject early in his career at Washington University and approached it with the mind of a biochemist rather than that of the "trained pediatrician" of those days. Because of his scanty clinical training and background he was not inhibited by tradition and such dogma as "percentage feeding," nor by the many fads and fancies in regard to the subject which were based on theory rather than fact. As Jeans expressed it, "He replaced empiricisms with scientific fundamentals."

His philosophy of infant feeding is well stated in the introduction to the First Edition of his text on *Infant Nutrition* published in 1930:

"With a thorough knowledge of the nutritional needs of the infant and of the means by which it is possible to meet those needs, the practitioner is in a position to appraise the value of any given type of feeding. If a formula or type of food is one which meets fully the requirements and at the same time is one which is capable of utilization without harm to the body, it will be successful. If it fails to meet any one of the essential requirements, it will fail as a whole. If these self-evident facts were more fully appreciated, there would be few-

er failures due to the feeding of well-balanced digestible formulas in insufficient amounts to meet the minimal energy requirements or the feeding of mixtures meeting the calorie demands but deficient in such essential elements as protein or some of the vitamins.

"There are numerous ways in which infants may be fed successfully, and there is no one method of feeding or type of food which is to be recommended to the exclusion of others; on the other hand, there is no reason to resort to the use of complicated formulas or expensive proprietary preparations when the particular requirements in the individual case may be fully met by simpler means."

It was this philosophy of infant feeding that led to what came to be known as the "Marriott method." Independently but coincidentally with that brilliant clinician Joseph Brennemann of Chicago, he became convinced that "evaporated milk" was the best practical source of a safe milk for the infant. Many of the pediatricians of that time had gone through the era of "condensed" milk with its excessive sugar content, and held a deep distrust of a "canned milk" as the basis of the infant's formula. To the evaporated milk diluted with water, which met the protein requirements, Marriott added lactic acid, which obviated the tedious bacterial souring of milk to obtain an acid milk. Acid milks were nothing new, as buttermilk has been used in infant feeding for several hundred years, and cultured acid milks were used by pediatricians for "difficult" cases, but their use was uncommon rather than routine because of the difficulty of preparation. Marriott's explanation of the value of the added acid was that it neutralized a portion of the buffer substances present, and thus the degree of acidity in the stomach approximated that found when human milk is fed. To this simple basis he added enough carbohydrate to make the caloric intake meet the energy needs. The use of Karo for the carbohydrate, which many have considered an essential part of the "Marriott method," was not fundamental. Its use came about in an interesting way. Cane sugar was the chief source of carbohydrate added to the infant's formula, especially in the infant welfare stations, although in private practice many pediatricians used and preferred some of the more expensive proprietary forms of sugar. In the early twenties cane sugar for a time became scarce and expensive (up to 20 cents a pound, which would be equivalent to 40 cents a pound today) and, further, in some localities a doctor's prescription was required to get a pound for the infant's formula. At that time we were expanding the infant welfare centers in St. Louis, and sugar, because of its cost and scarcity, became a problem. We talked the problem over several times and one day Marriott came up with the idea that Karo, which was inexpensive and in abundant supply, might be used for the carbohydrate in the formula, as it contained 50 per cent dextrin, 30 per cent maltose, and 10 per cent each of dextrose and sucrose—a clinical composition quite similar to some of the more expensive forms of carbohydrate in successful use. Clinical trials soon showed it was a satisfactory substitute for cane sugar. It is an excellent example of how Marriott applied his chemical knowledge to an important practical clinical problem. One wonders if the method would have been accepted so rapidly and so widely

if it had been put forward by someone without Marriott's prestige. It might, perhaps, have remained unnoticed had it been developed by anyone else. It is an unanswerable speculation.

His well-known text, *Infant Nutrition*, was based on mimeographed notes he had prepared for distribution with his lectures to undergraduate students, and particularly to those taking the postgraduate course held each spring and fall at the St. Louis Children's Hospital. Philip C. Jeans had an active part in the preparation of some of the "notes" which eventually became incorporated in the text, and, following Marriott's death, took over the third and fourth editions of the text. The first edition in 1930 ran into three printings and over 7,000 copies were sold. In no time at all it became the standard text on infant nutrition in many medical schools.

When one looks over the programs of pediatric meetings thirty years ago, and compares them with the program of a meeting today, where a paper on infant feeding is almost a curiosity, one realizes that there is an element of truth in a remark of Marriott which Park quotes: "Infant feeding, there is nothing to it." There is still "something" to it, but due in part to Marriott's teachings it is no longer the chief problem of pediatrics as it was some forty to fifty years ago.

He was an able teacher, particularly when it was a subject such as infant feeding from the didactic standpoint, or some disease as diabetes or nephritis where pathologic physiology was involved. He could present complicated chemical problems in a simple and clear way which enabled the listener to grasp the subject. Undoubtedly his long experience in teaching biochemistry to first-year medical students stood him in good stead. The purely clinical teaching he left largely to his associates, who included, among others, Jeans, T. C. Hempleman who was perhaps the best clinician in the group, and Hugh McCulloch. Later Jean Cooke and Hartmann took over the chief load. One thing that always amused us was his dislike to have anything to do with the "contagious disease" wards. He avoided entering them if it were possible. Perhaps he had a premonition of his low resistance to infectious diseases.

There was always a respect for intellectual integrity in Marriott's clinic. Every member of the staff taught what he believed. In the postgraduate course, Marriott gave the lectures on infant feeding and it fell to me to give the practical clinical instruction in the infant welfare clinic. At the end of one clinic one of the physicians taking the course remarked in regard to some point, "You teach one thing and Dr. Marriott another." My reply was that we disagreed on a number of things, but Dr. Marriott, the head of the department, felt that everyone must teach what he believed and practiced, and there was no rule, as existed in some departments of pediatrics in other schools, which forced everyone to teach what the "Chief" believed, or lose his position. Perhaps it may have been confusing to the student at times, but it was a matter of intellectual honesty in which Marriott firmly believed.

In 1923 Marriott became Dean of the Medical School at Washington University, a position he held until he left for San Francisco in 1936 to become Dean and professor of research medicine at the University of Cali-

fornia. My feeling at that time was that it was a tragedy for pediatries, as after becoming Dean his scientific contributions became of relatively little importance as well as infrequent. He liked the position of Dean and its administrative duties, and his active mind soon became largely taken up with problems of medical education. The load of two major jobs was too much for him from the standpoint of time for pediatric work and, as a result, his pediatric contributions suffered. The tragedy lay in the fact that while many men had as good or even better administrative ability, there were very, very few with Marriott's brilliant scientific mind and ability.

Looking back on it from the vantage of years, I have come to the view that his switching his main interest and work was due to the fact that he began to feel there were no further major contributions in pediatries for him to make. He had not gone into pediatries because of an interest in the clinical side of pediatries, or an interest in children, but by chance and because it gave him the opportunity to study and investigate clinical problems from a biochemical standpoint. Had he gone into internal medicine, as he originally wanted to do, he would have become as outstanding a figure in internal medicine as he became in pediatries. Once in the early thirties, when we were returning from a meeting of the American Pediatric Society, he stated, in answer to a remark that he had not said a word at the meeting, that he felt "out of the pediatric picture." In a space of only ten years Marriott's brilliant scientific contributions to pediatries had made him an outstanding figure, and this was his attitude only a few years later. He had, I believe, lost interest in pediatries and sought other fields to conquer. In discussing with me the move to California he said it would give him the opportunity to work on some adult problems he had in mind, and added that "you have known of this interest and desire for many years." Unfortunately he was never able to accomplish this desire. In 1933, shortly after starting on a vacation, he came down with a mysterious disease that turned out to be the "St. Louis" type of virus encephalitis, which became epidemic in St. Louis later that summer. He never fully recovered as it left residuals which impaired his health. He had been in San Francisco only a few months when he developed a fatal streptococcic infection as a complication of an inflammatory abdominal condition.

"What sort of person is Marriott?" This was a frequent question asked during his life, and, in the past tense, ever since his death. The answer will depend to some extent on the person who is asked the question. Most of us who knew him well and were associated with him will agree with Alexis Hartmann, who shortly after Marriott's death spoke of his qualities as having ". . . won for him the love of all of us, who as long as we live will mourn his passing and cherish his memory." There were some of his associates who respected him, but for whom the word "love" cannot be used. He was not an easy person to know, and during his life other able men prominent in pediatries complained they could not get close to him. He was fundamentally a shy person, and had a rather terse, abrupt way of speaking which was likely to give a first impression that he was stand-

offish and had a feeling of superiority. He was a very simple person in most ways and not at all what is termed "high hat." The fame and position in medicine that he reached in such a rapid and rather explosive manner never went to his head. There was never any real change between the Marriott I first knew, when he was the "third stringer" in the department of biochemistry, and the Marriott when he became an outstanding figure in the pediatric world.

He lived for his work, and his only real outside interest was in his family and home. His tastes were the simplest. He did not play golf or tennis, nor did he fish or hunt. He enjoyed swimming and hiking and at times wore out younger members of his staff who tried to keep up with the "Chief." He had no interest in music, and I doubt if he ever set foot in an art museum of his own free will. His reading was largely technical. About the only relaxation he liked to take and seemed to enjoy was to drive into the nearby Ozarks with some of the staff. Occasionally he would start for the Rockies in his car, drive until he became tired, go to sleep along the roadside or under a haystack until rested, and then start on driving again. Marriott did not like the cold mornings in the mountains, and on an early start would put on about five layers of clothing which he would gradually discard. Alexis Hartmann, who accompanied him on some of these trips, tells of one of the few occasions in their long association that he saw McKim get angry. Shortly after starting out early one morning they had a "flat" and Marriott had to unwrap to change the tire. Another of his recollections of the trips was Marriott's appetite. Two breakfasts and two luncheons were not unusual. Marriott disliked heat as well as cold, and in hot July weather in St. Louis would walk down the corridor to the interns' quarters a half dozen times a day to take a shower bath.

Once he bought an old secondhand car in St. Louis and started for California where he was to give some lectures. The pistons were so worn out the engine scarcely had power to get the car over the mountains. "But," as he related it to me, "I filled it up with heavy oil for a demonstration and sold it in California for more than I paid for it in St. Louis." This brings out another side of Marriott, and one which does not fit in with the usual conception of a scientist. He had a fine business sense and could drive a very good "deal," which he did at times. He never lived in the clouds when it came to a real estate or business transaction, or some financial matter he had to decide upon as Dean of the medical school. He was as practical here as he was in his scientific work and in applying biochemistry to clinical problems.

He was not an unerring judge of men or their ability. Some of his close associates at times were men of mediocre medical ability. Although not entirely his fault, there were some unfortunate appointments to the medical school faculty during his deanship, and at least one of these developed into a serious problem for him.

Marriott physically was a large but active man. He had a soft, rather high-pitched voice which one did not expect from his physique. He had an excellent sense of humor and enjoyed it when the joke was on him as well as on someone else. He

was the opposite of quick tempered, and anger was rarely seen. His mind was always active and at work, even at times when most men would be mentally relaxed. It was always something "medical" on his mind. Several times we motored two to three hundred miles together to attend a medical meeting and the conversation practically never got away from shop. He would come up with the most ingenious ideas and schemes imaginable that he had been mulling over.

Marriott was neither an introvert nor an extrovert, although he had a good sense of showmanship which helped in putting over his ideas. He enjoyed "barnstorming." One of the attributes that stood out was confidence. He had confidence in himself, his work, and his plans. It was not egotism as some felt who did not know him well. Another characteristic was his optimism. Everything was not only possible, but easy. He had a tremendous enthusiasm for everything he undertook which was contagious to those about him. He would become intensely preoccupied at times and walk by everyone without the slightest sign of recognition. Quite naturally those who did not know him well felt hurt on these occasions and looked upon him as "high hat." Those of us who knew him well would never give it a second thought, or if we wanted to speak to him about something would simply yell at him and he would "come to" and be perfectly affable. He was completely generous to his colleagues, as well as approachable to talk over their problems both medical and personal. Another characteristic was his freedom from worrying about what might happen. "Wait until it happens before you begin to worry," was in one way or another a frequently expressed philosophy toward life and his medical work.

In his political philosophy he was a pacifist and an isolationist. During the few years before the United States entered World War I, when he was at Hopkins, some of his associates considered him a "militant pacifist." For him there were no moral questions involved, and he regarded the Germans, French, and British as being in the same category—"fools." His encounters with Howland on the subject were said at times to be most amusing to the staff, as Howland's feelings and attitude were quite to the contrary. Throughout his life he seemingly had little interest in what went on in the world at large. His horizon was almost completely limited to the immediate environment in which he lived and worked.

Marriott's pediatric career was unique. It was built upon a sound foundation, a normal, well-balanced personality, and an unusually brilliant, imaginative, and practical mind which was wholly taken up with his work. The first period of his medical career was the decade 1904 to 1914 which was spent in acquiring a sound knowledge of biochemistry under such able men as Wolf and Shaffer. Then came the second, an almost fantastic and unbelievable period, lasting only another decade, 1914 to roughly 1924, in which the obscure biochemist became an outstanding figure in the pediatric and medical world. It was fortunate that the first three of these years were in association with the able clinician Howland, who had come to the realization that further progress in pediatrics must be built on integration with the basic sciences. Howland was without

adequate training in these fields and Marriott was without clinical training and experience. The two in their work together fused into a complete whole, and it is useless to try and separate their joint work into components. Then for seven years Marriott was on his own, and important and lasting contributions continued until the third period of Marriott's career, beginning around 1923-1924, when he took on the administrative duties of Dean of the Medical School. His interest in and contributions to pediatrics waned. It is given to few men in a lifetime of hard work and endeavor to make one important contribution to medicine. Marriott in the short span of ten years made four. Perhaps he was as well summed up as can be done by a remark of Howland, which Park relates: "One time when in a moment of discouragement I was discussing my own future with Howland, I made a reference to Marriott, which might have been construed as a comparison. Howland said, 'You must not be governed by Marriott. He is an exception.' "

Borden Veeder.

Comments on Current Literature

CIRCULATORY ADAPTATIONS TO BIRTH

THE July, 1955, issue of the *American Journal of Obstetrics and Gynecology*[1] carries a most informative article by S. R. M. Reynolds, Ph.D., embryologist and physiologist, Carnegie Institution of Washington. This report, presented before the Chicago and the Washington, D. C., Gynecological Societies is entitled "Circulatory Adaptations to Birth and their Clinical Implications."

The author points out that, in order to understand the circulatory changes occurring at birth, knowledge concerning the hemodynamics of the fetal circulation is essential. Three features distinguish the fetal circulation from that of the adult: "(1) the ductus arteriosus which connects the pulmonary trunk with the descending aorta, (2) the foramen ovale which permits 'fresh' blood returning from the placenta to pass to the left side of the heart and thence to the forepart of the fetus, and (3) the ductus venosus, a structure that permits blood to by-pass the fetal liver in returning from the placenta to the fetal heart."[1] While these features are primary in the fetal circulation, "they by no means reveal the peculiar or special physiological nature which marks it as different from that of the adult."[1] There are three other "important respects in which the fetal circulation differs from that of the postnatal organism . . . : (a) the gradient of blood pressure throughout the system; (b) the speed and volume of blood moved through certain parts of the system, and (c) the pattern of nervous control of the fetal circulation."[1]

Reynolds[1, 2] has made a study of fetal hemodynamics in animals, particularly from the standpoint of pressure gradients. After birth the level of blood pressure is high throughout the arteries, with the sharpest fall of pressure in the arterioles. The pressure in the capillaries is only a little above the pressure exerted by the osmotic pressure of the plasma protein. Pressure drop occurs in placental arterioles, but in the capillary network of the placenta, including the fetal villi, pressure is high—about 40 to 50 mm. of mercury. In the umbilical vein the pressure is about 35 to 45 mm. of mercury, a level higher than that of any other vein in the body. These are findings recorded in the circulatory system of the fetal sheep. In a study of fetal circulation in rhesus monkeys it was noted that, as intrauterine pressure rises when the uterus contracts, the fetal blood pressure rises also, and to the same extent. Thus the fetus is able to maintain a relatively constant differential of pressure between the inside and the outside of its vascular system.

The placenta is drained by the umbilical vein to the inferior vena cava; the route may be through the ductus venosus, or by way of the hepatic circulation. From a study of rapid radiographs, the author was able to show that there is a sphincter mechanism at the beginning of the ductus venosus which can close within one-third of a second. This sphincter is innervated by the vagus nerve. Reynolds believes that the ductus venosus closes, by means of this sphincter, to

divert to the liver blood which is returning in high volume, in order that sudden overloading of the fetal heart may be prevented.

High pressure in the umbilical vein maintains a high pressure throughout the cord and the placenta, a dynamic situation which makes the cord "during life an erectile structure." Distention keeps the arteries and veins large; slight pressure drop can occur due to friction, while at the same time the volume flow of blood can be large. Nature protects the fetal heart also by dynamic volume flow of blood throughout the organism. Fetal cardiac output permits 75 per cent of the blood to go to the caudal end of the fetus, 15 per cent to the head, and 10 per cent to the fetal lungs. About two-thirds of the aortic flow, or 50 per cent of the cardiac output, passes to the placenta. The determining factor in placental blood flow is not the length nor the tortuosity of the cord, but rather the fetal cardiac output, itself. The velocity of blood flow is fastest in the descending aorta and slowest in the head end of the fetus.

Of special interest from the obstetric and pediatric point of view are physiologic factors influencing reflex control of the fetal heart, and the absence of vagal tone in the resting fetus. Experimental studies of fetal hemodynamics in animals help to elucidate the type of physiologic disturbance which can lead to fetal distress. Obstetricians have emphasized the fact that marked or prolonged slowing of the fetal heartbeat during labor indicates fetal distress. Barcroft[3] demonstrated that slowing of the fetal heart is not attributable to anoxia alone, and explained that the reflex can be associated with a rise in arterial blood pressure when the umbilical cord is clamped, reasoning that the reflex is depressive. Experiments by Reynolds have shown, however, that the same reflex slowing occurs if only the umbilical veins are occluded, and there is no increase in blood pressure; Reynolds attributes fetal bradycardia to reflex action arising from the large veins of the fetus. In fetal circulatory distress and anoxia, when prolonged tachycardia follows bradycardia, the cardio-accelerating stimulus arises from mass stimulation of the sympathetic system, and the prognosis for the infant is considered to be poor. This sign means that the fetus already has been pushed to the last of its physiologic reserves. In the resting fetus there is no vagal tone; when the vagus nerves are cut, the heart slows down instead of speeding up as in postnatal animals. Yet when the peripheral ends of the cut vagi are stimulated, the heart stops, indicating that the fetal vagus is inhibitory in action. Vagal tone can be induced in response to stress. Thus in the resting fetus, the nerve connections are intact: by analogy, "the wires are up, the connections made, but the line is not busy."[1] Fetal circulatory distress induces vagal tone, and sets off these reflex mechanisms.

At the time of birth the shift from fetal to postnatal circulation is sudden; extensive fall in blood pressure occurs throughout the entire animal during the initial moments following birth. This observation, demonstrable in animals, and verified in the human fetus, was first recorded clinically by the German physician, Schultze, in 1871. At the time of birth, pulmonary circulation speeds up, as does that to the head; thus, circulation is now rapid where it had been slow prenatally, and slow where it was rapid. The capacity of the fetus to respond to stress as these adjustments take place depends upon adequate sympathetic response mechanisms. In the opinion of the author, adrenalytic substances to shorten labor are contraindicated since they have an adverse effect upon the fetus, and may lead to delayed postnatal pathology, such as subarachnoid hemorrhage. The author likewise emphasizes the advisability of holding the infant below

the level of the placenta, in order to give the benefit to the infant of additional blood by gravity. Infants who gain this added blood during the first few minutes following birth seem to do better in the perinatal period than those infants who do not have this advantage.

Studies such as these being conducted by Reynolds which concern fetal hemodynamics, pressure gradients, forces of blood flow in the umbilical cord, etc., have important "clinical overtones," and will foster a better understanding of situations associated with the incident of birth, including fetal distress.

RUSSELL J. BLATTNER.

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2. Reynolds, S. R. M.: Hemodynamic Characteristics of the Fetal Circulation, Am. J. Obst. & Gynec. **68**: 69, 1954 (Festschrift in Honor of Dr. Geo. W. Kosmak).
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News and Notes

The annual meeting of the **American Academy of Pediatrics** was held in Chicago Oct. 3 to 6, 1955. Dr. Harry Bakwin was installed as President and Dr. Edgar E. Martmer, Vice President and President Elect. The annual meeting in 1956 will be held in New York.

At a meeting of the Section on Allergy of the **American Academy of Pediatrics,** held at the Palmer House in Chicago, Ill., a scroll was presented to Dr. Bret Ratner of New York City by Dr. Jerome Glaser of Rochester, N. Y., chairman, as an expression of gratitude and appreciation of the Section for his untiring services in advancing pediatric allergy.

In June the Committee on the Control of Infectious Diseases of the **American Academy of Pediatrics** issued a statement advising the use of the Salk vaccine for polio be stopped until more detailed information became available. At the last Academy meeting the Committee issued the following statement:

"In view of the information made available to the Committee by the United States Public Health Service, and by others, which indicates a trend in favor of protection against paralytic poliomyelitis in recipients of poliomyelitis vaccine; in view of recent improvements in production, and in the control and testing of such vaccine; the Committee approves the resumption of vaccination against poliomyelitis in the priority age groups established by responsible authorities at state and territorial levels.

"The Committee commends the continuing efforts to develop further improvements in poliomyelitis vaccines and their methods of production and control; and the continuation of careful surveillance."

The **Southwestern Poliomyelitis Respiratory Center** at Houston, Texas, will give a course on the *Clinical Management of Poliomyelitis* on Feb. 27, 28, 29 and March 1 and 2, 1956. The course is sponsored by the Center, Baylor University College of Medicine, and Jefferson Davis Hospital. Detailed information as to the course may be obtained from Dr. William A. Spencer, Jefferson Davis Hospital, 1801 Buffalo Drive, Houston 3, Texas.

Books

Communicable Diseases. Franklin H. Top, M.D., and Collaborators, St. Louis, 1955, The C. V. Mosby Co., 1208 pages. Price $18.50.

It is suggested in the preface that a new book on the clinical aspects of communicable disease may be unnecessary. Specific preventive methods and specific therapy have brought us a long way from the pest house in the management of contagious disease, and modern concepts are concerned more with the care of the infected individual than with the protection of the community. Nevertheless careful description of the course and control of these conditions of decreasing frequency is invaluable for the instruction of medical students—which must now perforce be largely didactic—and for the guidance of practitioners. Much that is included in this volume is best written by men like Dr. Top whose experience spans the course of untreated disease in the preantibiotic era and the present period of greatly augmented therapy.

It is understandable that some of the discussion should be reminiscent and almost nostalgic. Many things have changed but old problems remain even though on a lesser scale.

The preliminary discussion of infection and immunity is excellent. The index is adequate, the illustrations are unusually good, and there is a very extensive and useful bibliography. Chapters on sulfonamides and antibiotics are succinct and informative. Discussion of home and hospital nursing care appears to be somewhat cumbersome and reflects the difficulty of translating earlier practice into modern methodology. A number of new chapters have been added which are written by outstanding experts in specific infectious diseases.

The wealth of documentation and the inclusion of so many different methods of treatment may confuse the student or the practitioner in his own choice of procedure. The author throughout might profitably have expressed his opinions derived from a rich experience about acceptable measures.

Certain subjects are curiously omitted from more than passing consideration and the experienced reader may encounter some things with which he may not be in complete agreement. To mention only a few of these:

(1) Immunizing agents are proposed in generally larger volume for total dosage than is usually recommended—almost all of these are now recommended in 0.5 c.c. dosage.

(2) BCG receives scant attention and no explicit advice about its usefulness and methods of employment.

(3) Intubation is proposed as preferable to tracheotomy in the treatment of laryngeal diphtheria—neglecting the idea that in inexperienced hands (and where is one to get experience) tracheotomy may be much safer.

(4) Tracheotomy is only briefly mentioned in the care of bulbar poliomyelitis although other authorities have considered this a matter of vital importance and have devoted considerable discussion to it.

(5) Cortisone and hydrocortisone are mentioned cursorily in connection with hepatitis and rheumatic fever, but the use of these drugs in serum disease and similar allergic states, in fulminant meningococcemia, in mumps orchitis, etc., have been entirely omitted.

(6) von Economo's encephalitis is discussed as though it were now a matter of present common experience.

(7) It is recommended that scarlet fever and manifest streptococcal disease of moderate severity may be treated by purely symptomatic measures.

(8) In numerous instances antisera which are now practically nonprocurable are mentioned for therapeutic use.

(9) Roseola is given as a synonym for measles.

Most of the minor shortcomings of this book simply reflect obviously difficulties, with which a reviewer must be most sympathetic, of translating past experience into present practice. This book contains a vast amount of information but must be read with discrimination and will not serve as an explicit guide for the complete tyro in this field.

Sexual Precocity. Hugh Jolly, M.D., M.R.C.P., Springfield, Ill., 1955, Charles C Thomas, 276 pages. Price $6.75.

An interesting monograph, published simultaneously in England by Blackwell. There are many case reports of sexual precocity in medical literature, but few series of cases studied by one observer. Dr. Jolly has personally studied 69 cases in England which in large part were seen by physicians at the Great Ormond Street Hospital. Of these, 50 were female and only 19 male. About 50 per cent he classified as "constitutional" and considers them as due to some lesion of the hypothalmic area of the brain. Only 5 were due to brain tumor, and 9 to adrenal tumor or hyperfunction. In only one instance was a granulosa tumor of the ovary found. The first part of the monograph (92 pages) takes up classification, and a discussion of the various causes, questions of diagnosis, treatment, and prognosis. This is followed by an excellent and complete bibliography. The final section of about 150 pages takes up the individual report and study of each of the 69 cases. They are unusually well done with the important data included.

They contain a wealth of factual material, with numerous photographs and illustrations. It is an unusually fine study and report, and the monograph will undoubtedly become a "classic" on the subject.

Premature Infants, ed. 2. Ethel C. Dunham, M.D., New York, 1955, Hoeber-Harper, 459 pages. Price $8.00.

The "first edition" of Dr. Dunham's *Premature Infants* appeared in 1948 as a publication of the Children's Bureau. It has been completely revised and much rewritten, and now appears in regular book form. The author has been interested in the premature infant for many years. It is, however, not a text of personal experience in recent years, but rather a compilation based on the voluminous literature on the subject which has been published with the growing interest in prematurity. The book is divided into three parts. The first section takes up "general considerations" such as definition, incidence, causes and prevention. She accepts the unscientific definition of 2,500 grams or less at birth, which for practical reasons has been widely adopted. The second part takes up "clinical considerations" such as feeding, care, and abnormal conditions. Under abnormal conditions such subjects as the congenital malformation found in the various systems, birth injury, infection, retrolental fibroplasia are presented. A third section presents the public health considerations of prematurity such as deaths and death rates, and public health programs which now exist in many cities and states. In an appendix a number of charts and tables are presented which relate to the subject. The text reflects a tremendous amount of study and research, and will be a most useful book of reference.

Diseases of Infancy and Childhood, ed. 7. Wilfrid Sheldon, M.D., F.R.C.P., London, 1955, J. & A. Churchill (distributed in the U. S. A. by Little, Brown and Co., Boston), 804 pages.

Dr. Sheldon's text differs considerably from the large encyclopedic American texts as Nelson, or Holt and McIntosh. Its purpose is a practical book for the student or practitioner, rather than a work of reference with a complete discussion of every disease of infancy or childhood. It gives, however, a readable and satisfactory discussion of the conditions the practitioner encounters in his daily work. In this, the seventh edition, a number of chapters, as those on dehydration and prematurity, have been completely rewritten to keep abreast of recent developments. A number of new subjects are introduced, such as retrolental fibroplasia, galactosuria, and methemoglobinemia. The author has an easy style of writing which together with the size of the book makes it a text that can be held and read with comfort and pleasure.

Radiographic Atlas of Skeletal Development of the Knee. E. I. Pyle, Ph.D., and Normand L. Hoerr, M.D., Springfield, Ill., 1955, Charles C Thomas, 82 pages. Price $4.25.

This atlas of radiographic standards of reference is based on the work started nearly thirty years ago by the late T. Wingate Todd, Director of the Brush Foundation of the School of Medicine of Western Reserve. The earlier atlases of standards for the hand by Dr. Todd (1938), and hand and wrist by Drs. Greulich and Pyle (1950) have become standard texts of reference for skeletal maturation. The atlas presents 28 plates showing the skeletal development of the osseous structures of the knee at ages extending from early infancy to 18 years. Whether it will equal or replace the earlier standards of maturation for the hand and wrist in clinical use only time will tell. The printing and format are excellent and the plates clear.

Editor's Column

THE INFLUENCE OF DAVID EDSALL ON PEDIATRICS IN THE UNITED STATES

WITH quite another purpose in mind, the Editor recently picked up Volume 22 of the *Transactions of the American Pediatric Society* with the proceedings of the meeting in Washington in 1910. The president was David Edsall, an internist who became an outstanding leader in medical education. His address, given forty-five years ago, is so interesting and of such historical importance in the development of pediatrics in the United States that we are reprinting it below. The frank way in which he pointed out that pediatrics at that time was not living up to its possibilities, nor attracting men to enter the specialty, undoubtedly was not relished by some of the members of the Society who were the pediatric leaders at that time. His comparison with neurology is most interesting. Over the years the neurologist has largely been replaced by the neurological surgeon and the psychiatrist, while pediatrics has developed to the position which Edsall clearly saw should be attained. Since Edsall was primarily an internist with a secondary interest in pediatrics, as were many of the members of the pediatric society in its early days among whom Osler is a striking example, and his chief contribution to medicine was in the field of medical education, his name was never thought of as a subject for a "Pediatric Profile." Yet as one looks back on it, he undoubtedly had a greater influence on the developments which led to pediatrics as it exists today than some of the men who have been the subject of a Profile.

In 1909 and 1910, the year of his presidential address, Edsall was in the process of selecting the faculty for the medical school of Washington University which was being reorganized. His selections were Opie for pathology, Erlanger for physiology, Shaffer for biochemistry, Fred T. Murphy for surgery, while he himself was to head internal medicine and act as Dean. His thinking as expressed in the presidential address was that pediatrics was a major clinical subject and should be developed and organized equal in rank and importance to the other clinical subjects. To develop pediatrics on this basis, his selection was John Howland. To regard pediatrics in this way was unusual and a rather unique concept forty-five years ago. A few years later when he had left St. Louis to become Dean of the Harvard Medical School, one of his important moves in line with his concept of what pediatrics should be was to place Oscar Schloss of New York at the head of the department at Boston. The stories of Schloss and of Kenneth Blackfan who succeeded him at Boston have been told in their Profiles appearing in previous issues.

It is only when one considers the picture of pediatrics some fifty years ago, which is so frankly discussed in

Edsall's presidential address, and Edsall's "advanced ideas" of the changes that must be made if pediatrics was to attain the place he felt it should have in the medical curriculum, and to this add the steps he took to put his ideas into effect, that the importance of his influence is realized. While his scientific contributions to pediatrics were few, his contribution to its development makes him an important figure in its history.

Annual Address of the President, American Pediatric Society

May 3, 1910

The honor of serving for a year as the president of a group of men who are the highest representatives of their type of work in this country imposes upon one, as I conceive of it, the duty not merely of giving expression to complacent satisfaction, but also of devoting serious thought to the activities that may be open to the members of the Society as individuals or as a group by means of which they may still further advance the honorable standing of the work that is their chief concern in life. I am in a somewhat dangerous position in approaching this duty, for since my own work has come to be more largely outside the field of pediatrics than in it I may easily find myself presenting much the same appearance to you, the masters of pediatrics, as does the occasional maiden lady of philanthropic tendencies who would instruct mothers in suitable methods of training children. This is the more likely to be the case inasmuch as the attempt to urge new activities upon you involves not only the giving of unsought advice to those who are my superiors, but also it carries with it some suggestion of criticism in that it presupposes that I consider those things that have been done to be not yet sufficient. I recognize fully, however, that the things that I have to say cannot be considered exactly as advice, inasmuch as they are not my own thoughts alone, but in more or less the same form are uppermost in the minds of many others among you; and as to any flavor of criticism, this is due not to any lack of appreciation of what has been done, but to my feeling that in spite of all that has been done, proper dignity and regard are not yet generally accorded in this country to a subject which, it appears to me, is unquestionably the broadest and most complex and the most important division of general medicine, and proper standing will not be generally given to pediatrics in this country until better things are demanded for it by concerted action, and until also better things are in certain ways demanded of it by a large group of those who are the recognized leaders.

The dignity of any of the intellectual pursuits and the standing which it has in comparison with other things related to it are determined chiefly by two things. First of all, I think, by the opportunities offered those who teach the subject and by the manner in which these opportunities are made use of; and, second, by the extent to which those who follow that pursuit engage in matters that favorably affect the welfare not only of those individuals with whom they come in immediate contact, but rather of the pub-

lic at large. There are, it appears to me, ways in which greater usefulness could be secured for pediatries in both of these directions, and I believe that this society could rapidly, by the strength of carefully planned concerted action, greatly benefit the position of the work which it represents. It may appear to many of you somewhat out of place for me in addressing this society to devote most of my time to the question of the teaching of pediatries, but I think an apology for doing so is hardly necessary, partly because most of you are teachers, but also for a much broader reason than this; that is, it is undeniable that while teachers are far from constituting the only persons who have a determining influence upon the progress of their subject, they do have a most serious responsibility, not only because each of them has charge of a limited group of students, but much more because the methods they pursue chiefly determine the standards that are demanded in that subject, and even more important than this the teacher and his methods largely determine the quality of the future exponents of his subject, not merely through the actual information that he disseminates among students, but through his directly attracting a better or worse group of men into that subject as their lifework. The appearance of attractiveness of any subject to those who have not yet chosen their own special careers is most largely dependent upon the inherent interest in the subject itself and upon the breadth of view and enthusiasm of the teacher, but it is also, and often equally largely, dependent upon the halo which is given to the subject by the facilities which are provided for the teacher. It would be quite unsuitable and unnecessary for me to enter into a discussion of the worth of individuals who have charge of the teaching of pediatries in this country. I may dismiss this point by saying that among their number are men who have in certain ways no superiors in any part of the world. Concerning the provision made for their teaching, however, there are points which may justly be commented upon and not wholly favorably. I recently had a long discussion regarding medical education with one of the ablest educators in this country, and he told me most forcibly that an extended inspection of medical schools had convinced him that, considering its importance, pediatries is more inadequately provided for in most medical schools than any other important subject; and he frankly said that, considering the importance inherent in this subject, the conditions surrounding it are, in most places, shameful. He had investigated almost solely the best schools in this country. On the whole, I think we must agree with him in large part. I would hasten to say that his criticism was of the general group of schools that he had seen, not of all the individual schools and it does not apply in a number of instances where the system of teaching is relatively excellent, but at the same time I would say that nowhere is the provision made for the subject as good, even relatively, as that which is made for general medicine, general surgery and obstetries in a number of schools where these subjects are particularly well cared for. In fact, I may fairly say that a more directly comparable branch of work, neurology, has much more actual standing and recognition in the general group of our best medical schools than has pediatries; yet I

am personally convinced that the problems of infancy and early childhood are much more distinct from general medicine than are the problems of neurology, and consequently they more properly demand generous provision for instruction in them. They are at the present stage of our knowledge surely very much more important practically to the student.

An extremely striking, and, I believe, direct, result of the existing academic state of pediatries is that not only does the individual student receive in most places very inadequate training in the subject; even more significant than this is the fact that fewer able young men than might properly be expected to do so go into pediatries as contrasted with the number of such men that go into general medicine, and the number is less even than of those who go into neurology for at the present day we may certainly say that there are in this country at least twice the number of men of recognized distinction in neurology that there are in pediatries. The explanation for this is, I think, a reasonably patent one, and while the responsibility may properly be thrown in part upon the shoulders of the administrators of medical schools, it cannot be put wholly upon them and must be shared by pediatrists themselves.

It is a common, and, on the whole, a seemingly just, statement that the clinical branches have in this country in the past generation by no means kept pace with the medical sciences in their methods of teaching and investigation. I would especially point out here that in mentioning research as I have just done, I do it purposely, but not at all with the idea of pleading at the moment for research. I speak of it with the view of pleading for better general conditions in teaching, for it is, I believe, a well-recognized fact among educators that in most instances, unless a man does some kind of research more or less continuously, he does not make or, at any rate, does not long remain, a sound teacher, simply because the teacher must, in order to make the student search for knowledge, search diligently and continuously himself. Therefore, aside from the usefulness of knowledge gained through research, the kind of research work that is produced in any particular department is commonly accepted, and rightly so, as the best individual criterion of the degree of progressiveness that the teaching in that department will exhibit. Now during the past generation the medical sciences have been transformed from subjects that served chiefly to give those who taught them dignity and high position in closely related forms of practice, into subjects that of themselves furnish chosen and enviable careers to many of the best men in the profession. An inevitable and happy result of this has been that as they have assumed greater dignity and seriousness they have demanded and secured greater facilities and opportunities for teaching and research, and have in both these ways advanced with tremendous rapidity.

With the clinical branches no such fundamental change in point of view has occurred and none is possible; for continuous touch with practical matters is essential in the teaching of things that are necessarily practical, and therefore the teaching of clinical work cannot be wholly separated from practice. It has been impossible consequently to start wholly fresh in

these subjects upon an altogether new basis, and hence whatever change there has been in them has been in details rather than in general plan. Hence they still retain some of the casualness in teaching and research that the medical sciences cast off when they became purely academic subjects. Nevertheless there have been very significant forward steps in these branches and the occasional criticism of them that they have shown little, if any, progress is not by any means as well justified as it seems upon superficial consideration. I sincerely believe, however, that such a criticism is better justified when directed at pediatrics than when any other of the most important subjects in medicine is aimed at.

The chief essentials for making well trained men and for attracting first-class men into a subject as their own work are that the men shall do most of the work themselves and not merely be told about it; that they shall be made precise and critical in their methods, and that the craving for a sound understanding of the reasons for things be satisfied by having them clearly comprehend the relation between the fundamentals and the practical things. In all these ways general medicine and general surgery have been quite transformed within two decades in a large group of our best medical schools, and now, instead of the instruction being as it was earlier, almost wholly didactic and at best given in the form of clinical lectures to large classes, students do most of their work in wards and dispensaries, either in small groups or, still better, as individuals, acting practically as temporary assistant internes. Precision and a critical point of view are given them through the fact that purely observational methods, which always have an indeterminable amount of error associated with them, are supplemented by the employment of laboratory methods and instruments of precision, which not only give added information, but have the invaluable element of demanding accuracy and precision in their use and thus training into more precise habits. The craving for a fundamental understanding of facts that are rather baldly stated in textbooks is, in some places at least, being vigorously met by cooperative teaching between clinical and anatomical, physiological, pathological and other departments, or by much greater attention to these matters on the part of the clinical teacher, so that in either case the important bearings of the fundamentals upon clinical matters are brought prominently forth in immediate relation with the clinical teaching. In fact, a fair knowledge of physiology and pathology have become almost as important a part of a clinical teacher's training as is clinical experience itself, and that this is well recognized grows increasingly evident when one notes the character of a considerable group of appointments to important chairs in recent years. What I have said in regard to general medicine and general surgery is, to a large extent, true also of neurology, and to some extent of certain of the other specialties. There has likewise been movement in this regard in obstetrics and gynecology, though perhaps to a less degree than should have been the case. When we consider what has occurred in pediatrics I think we must admit that the changes have been much slighter and less distinctive. There has been decidedly less opportunity given the stu-

dent to work himself upon cases, and in many of the best schools he still sees children solely, or almost solely, through the eyes of the instructor. This is due to the fact that while the opportunities for actual work in wards and for bedside instruction in small classes have been growing everywhere in medicine and surgery, there are scarcely anywhere pediatric wards where students are freely permitted to do things themselves. The critical difference then is that while in medicine the student not only may, but in the best departments must, get extensive training in working out for himself the history, the physical examination, the examination of secretions, etc., in a series of cases, and often must, under the control of the instructor, plan out and follow the treatment, in pediatrics he usually does but a fraction of this; and as a necessary result cases are less frequently seen as problems to be worked out by the student himself than as problems which the instructor both points out to him and solves for him. Naturally this is less attractive to the best men. Healthy-minded persons enjoy eating more than they do being fed. Guidance by instructors is, of course, essential, but it must be guidance in the methods of attacking problems rather than in the actual solving of these problems, especially when the students reach their final year. Furthermore, unless they do the work themselves the lack of opportunity of bringing for themselves all the methods of precision to bear upon each case leads students into less exact methods and into less respect for the subject that they are dealing with. Again, as I said, I believe the serious-minded student takes a deep interest in any subject only when he has clear conceptions of its relations to those things on which it is based. This is to my mind the most important side of the matter that I am discussing, though not usually dwelt upon. It is, it seems to me, the crux of the whole situation in so far as the character and number of the men going into pediatries is concerned. So far as I can see, the propriety of studying pediatries as a special subject rather than as a mere part of general medicine is based essentially upon the fact that the infant and young child differ from adults enormously in degree and almost equally in kind, in susceptibility to infections, and especially in liability to derangements of nutrition. This constitutes a really profound difference, it appears to me, and gives much greater justification for specialization than any that is based upon the different organs of the body, for it demands of those who would enter into pediatries in a broad spirit very wide and subtle knowledge of the two types of work that are now, and probably will be for years to come, the most progressive and most productive lines of work in all medicine, but that are at the same time the most complex and difficult. I mean, on the one hand, infection, immunity and hygiene; on the other, the physiology and pathology of nutrition. In spite of the fact that these subjects are simply teeming with points that are of the most intense interest in acquiring sound and broad methods of practice, there are certainly scarcely any places where an attempt is made to bring out systematically and thoroughly their relation to clinical pediatries. So far as these subjects are at all broadly dwelt upon by clinical teachers, it is almost entirely in courses on general medicine not in

pediatries, though they are of even more intense interest practically in the latter. It is no wonder that in the eyes of many of the best students pediatries becomes the tail to the general medical kite, when the things most intimately related to it are largely neglected in direct relation to it, but are taught in practical relation to other clinical subjects. As I have intimated, however, I think that we may more justly compare the conditions surrounding pediatries to those in neurology rather than in those in general medicine, and, as I have said, to the disadvantage of pediatries. But the same explanation of the different status of the two may be used here: In every place where neurology occupies a conspicuous position in the curriculum and is highly regarded by students it will be found that the fundamentals are most intimately related to the clinical teaching, and there are special courses, usually directly associated with the clinical teaching, for broadening the student in his knowledge of the anatomy, physiology and pathology of the nervous system.

The manner in which this disadvantageous state of things can be overcome is, I think, indicated by what I have just said. Students should be taught in the pediatric department, or by the pediatric department in association with other departments—and not simply in some department entirely separate from pediatries—the important direct practical bearings of the study of the physiology and pathology of nutrition, of infection and immunity, and of all the fascinating things that fall under the name hygiene.

The overcoming of the difficulties in regard to the studying of cases can be accomplished only through demanding that the clinical work in pediatries shall be put on the same basis as that which is being struggled for, and increasingly secured in medicine and surgery; that is to say, there shall be pediatric hospitals, or at least wards of really adequate size where the department shall be absolutely at liberty not only to show cases, but to put the students at work in whatever way it is believed will lead to the best training for them. This is an old story in medicine now, and no teacher in clinical medicine is content with anything less whether he has obtained it or not; especially is he not content with a rotating service where the system changes with each change in the service, where he is thoroughly familiar with the cases only during his own term of service, where any investigation must be wholly fragmentary and interrupted and where everything is more or less dependent from day to day upon the goodwill of his colleagues and the hospital managers. Pediatrists have shown very much less evident activity in attempting to secure proper conditions of this kind and very much less apparent appreciation of the importance of such conditions than have those who are engaged in general medicine, surgery and obstetries, and yet such conditions are, I had almost said, more important than in the other subjects mentioned, for little details that one becomes familiar with only by direct contact with them, and not merely by hearing of them, are determining factors in successful diagnosis and treatment with children almost more than with adults. Nowhere in this country are the conditions as good yet as they are in a number of places in the other subjects mentioned. In two schools there will soon be depart-

ments with model systems even better than the best that now exist, where the head will have entire authority in a good hospital, will have the academic dignity and the salary that go with high ability and wide training, and will have the power to plan his teaching in whatever ways appears most efficient without regard to the fancies of the hospital managers; and where in return for these privileges it will be expected of him that he shall keep himself thoroughly trained, not only in pediatries itself, but in the lines of work that it most intimately touches upon, and that he shall not only disseminate knowledge, but contribute to it, and train others to do the same. Certain of our most distinguished members have previously established eras in pediatries in America, and one member, who is still stimulating us to higher ideals and better methods, is the veritable parent of the subject in this country. I anticipate that these new clinics will inaugurate another new era partly through the standards that they will set and that others must follow, but even more largely through the considerable group of highly trained men that the facilities provided in them will almost certainly lead into pediatries as their lifework.

Until conditions such as those that I have been discussing are generally recognized as the standard that is being striven for, I do not believe pediatries will assume the station that it deserves in comparison with other divisions of medicine. Certainly dependence upon hospitals that are not thoroughly controlled through either ownership by the school or firm affiliation with it is an anomaly that is almost as damaging to the best ideals in medical education at the present stage of things as was the money-getting spirit of the teaching in the days of the old proprietary schools. It appears to me that this society could wisely take upon itself a serious consideration of the manner in which teaching and investigation in this branch may be benefited, and then by both individual and concerted action further whatever appears to be wise. It is beyond question that acting as a group and with the power that comes through the name of the American Pediatric Society much could be rapidly accomplished. At this very time I know that a committee is preparing a report to one of our sister societies on the question of securing freer use of clinical material by students and better teaching facilities in other ways in the subject which that society represents. An authoritative report of this kind which will be recognized as representing not the ambitious desires of one man, but the earnest opinion of a commanding group of men, will have much force in bettering things if wisely used; indeed, I know of one or two schools that, knowing what this report recommends and desiring not to be found wanting when it appears, have already anticipated it and have advanced their standards to the point which it will demand.

In the earlier part of my address I spoke of the relation of men to the public at large as one of the main ways of being useful and one of the chief ways by which their usefulness is judged. There are a variety of ways in which this society could, I believe, act as the center for the dissemination of education, and particularly as the means through which knowledge could be acquired by us and by others interested in the subject in a consider-

able number of things which bear intimately upon the subject of pediatries and which are of great importance to the public at large, but in which reliable information is difficult for every one of us to secure. Some of these were discussed to a slight extent at last year's meeting. The conditions in children's schools could probably be led into a much better state than they are now were there some sort of conference between important educators in these schools and representative pediatrists such as might be furnished by a committee of this society. Industrial conditions among children have deeply interested me in connection with industrial conditions in general, and there is no question that they need study by persons of calm and judicial temperament so that we may be informed upon the actual state of affairs by those whose opinions would be more reliable than those of perhaps overenthusiastic philanthropists. I do not agree with the view expressed last year that such questions are outside the realm of research and are rather matters of mere medico-legal interest. The more I have studied occupational disorders the more fruitful a field of research I have found them. There are a number of other important question relating to other parts of hygiene in which I believe this society could take a useful part and a part that would in no way interfere with its other activities; and it could thereby cover subjects which are at present not particularly cared for by any other group of qualified persons, and concerning some of which it is now wellnigh impossible to get any satisfactory information or to offer reliable advice. Such matters I cannot take up *in extenso*, as I have already consumed an undue amount of time. I should be very glad, however, to see members of the society, or the society as a whole, consider the relation of the society to such matters, and either proceed to take some active part in them or condemn any such plan definitely.

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EDITED BY

A. B. SCHWARTZ, M.D.

From the Department of Pediatrics, Marquette University, School of Medicine, Milwaukee, Wis.

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EDITED BY

A. B. SCHWARTZ, M.D.

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EDITED BY

A. B. SCHWARTZ, M.D.

From the Department of Pediatrics, Marquette University, School of Medicine, Milwaukee, Wis.

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EDITED BY

A. B. SCHWARTZ, M.D.

From the Department of Pediatrics, Marquette University School of Medicine Milwaukee, Wis.

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EDITED BY

A. B. SCHWARTZ

From the Department of Pediatrics, Marquette University School of Medicine Milwaukee, Wis.

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EDITED BY

A. B. SCHWARTZ, M.D.

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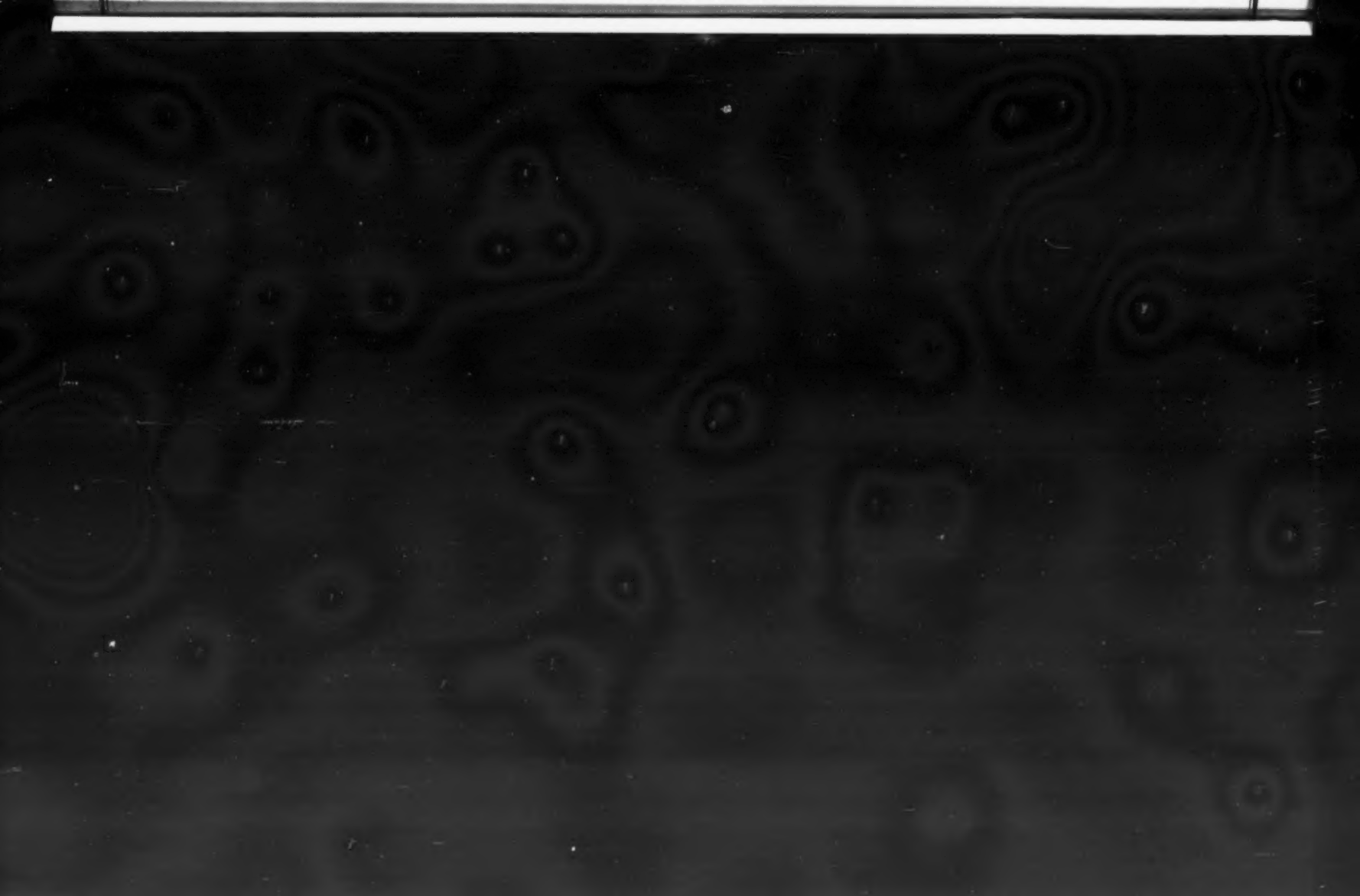

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A MONTHLY JOURNAL DEVOTED TO THE PROBLEMS AND DISEASES OF INFANCY AND CHILDHOOD

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- **simple to prepare and use in hospital or home**

Lytren is supplied in 8 oz. cans, with special measure enclosed.

References: (1) Conn, H. F.: Current Therapy, Philadelphia, Saunders, 1954, pp. 338-343. (2) Darrow, D. C.: Mod. Med. 22: 115-116, 1954; personal communications. (3) Harrison, H. E.: Pediat. Clin. North America, 1954, pp. 335-348; personal communications. (4) Cooke, R. E.: Personal communications.

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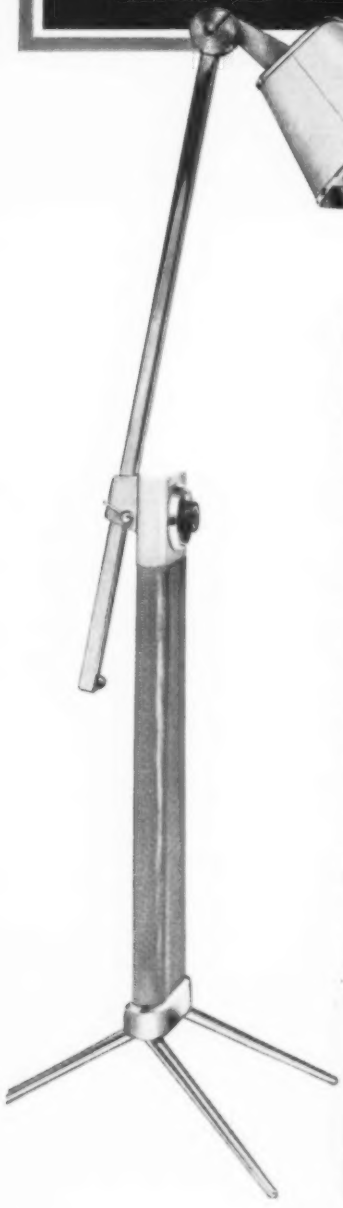

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Reference to RHINALGAN:

1. Van Alyea, O. E., and Donnelly, W. A.: E.E.N.&T. Monthly, 31, Nov. 1952.
2. Fox, S. L.: AMA Arch. Otolaryn., 53, 607-609, 1951.
3. Molomut, N., and Harber, A.: N.Y. Phys., 34, 14-18, 1950.
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6. Browd, Victor L.: Rehabilitation of Hearing, 1950.
7. Kugelmass, I. Newton: Handbook of the Common Acute Infectious Diseases, 1949.

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1. Allen, Jr., F. P.: A New Approach in the Therapy of Enterobius (Pinworms), J. Pediatrics, 46:155-157 (Feb.) 1955

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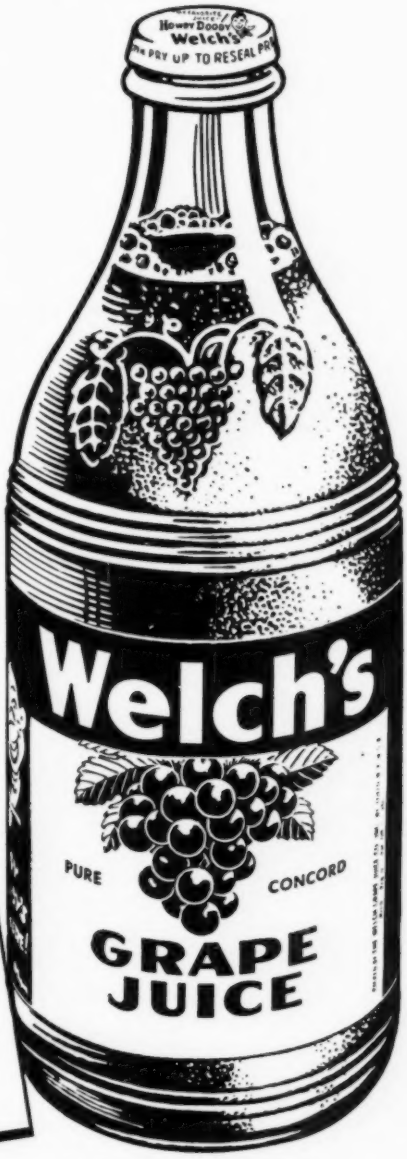
*Grape Juice in Infant Feeding by Alfred J. Vignec, M.D., and Samuel Weinberg, M.D., *The Journal of Pediatrics,* March, 1954.



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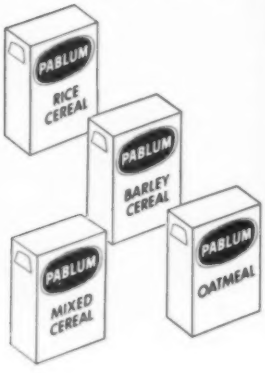

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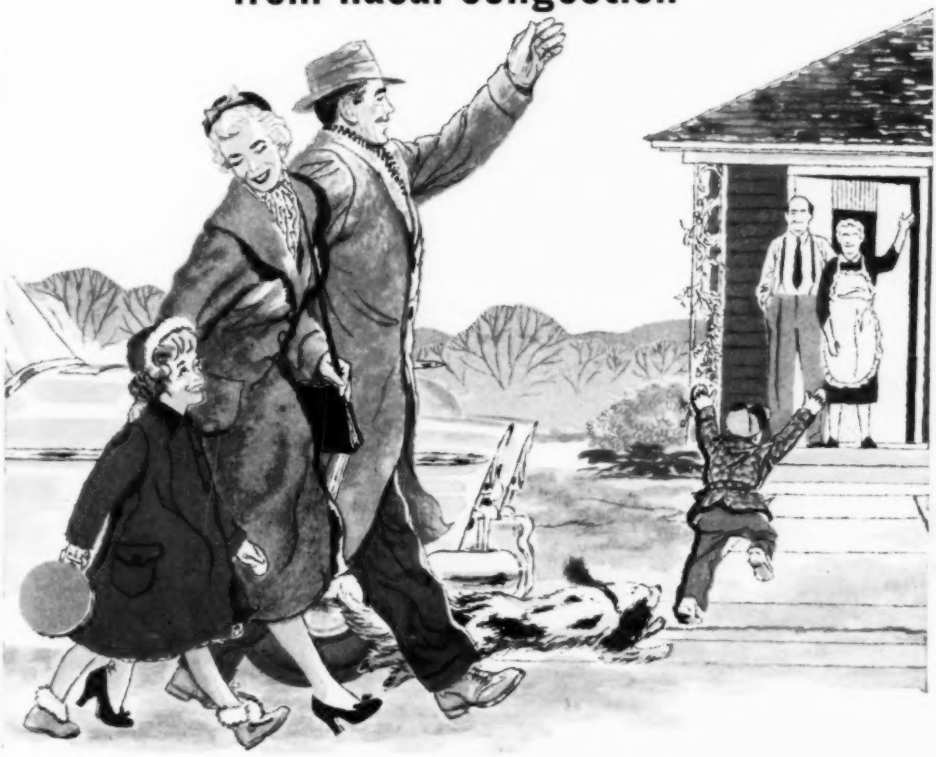

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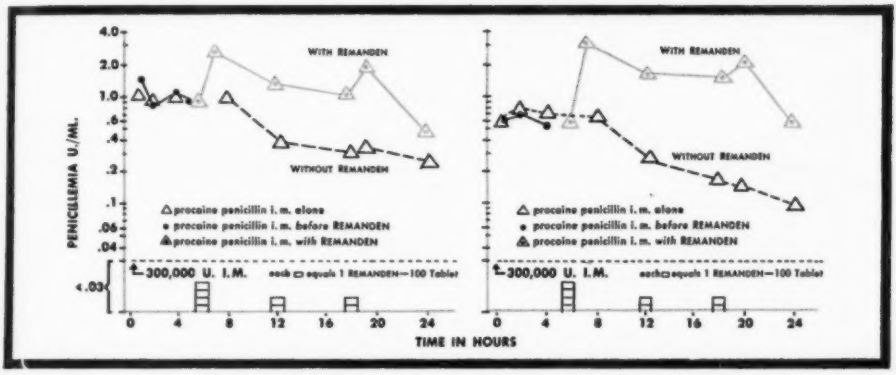
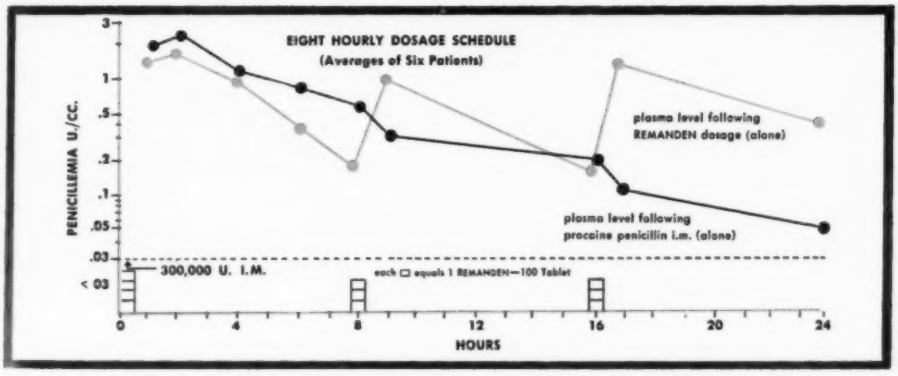


REMANDEN raises and prolongs i.m. plasma penicillin levels.[1]



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References: 1. Scientific Exhibit, Norristown State Hospital: Data to be published. 2. Antibiotics and Chemotherapy 2:555, 1952.

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SUPPLIED: In bottles of 3 fl.oz. and 16 fl.oz., through all pharmacies.

1. Bradley, J. E., et al.: J. Pediat. 38:41, 1951.

Literature and sample on request

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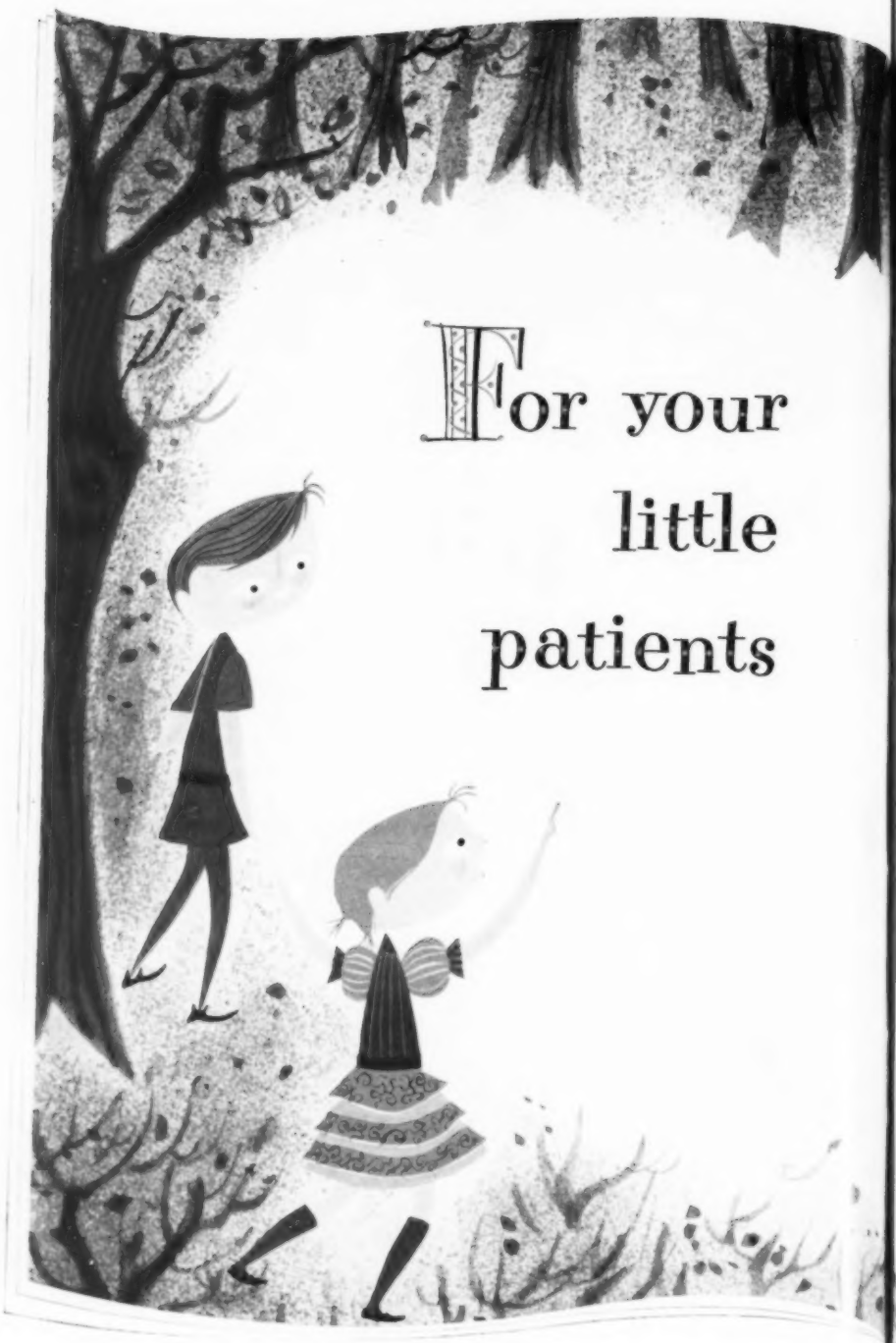
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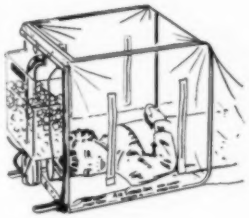
4. Refilling of water supply	Outside	Inside	Inside	Inside	Outside
5. Mist apparatus integral part of tent	Yes	No	Yes	No	No
6. Storage compactness	Yes	No	No	No	No



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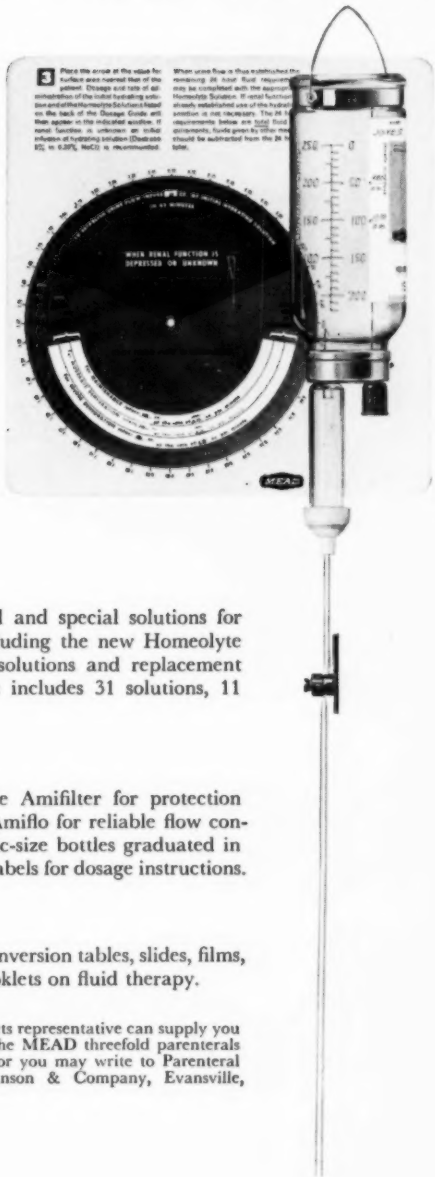
only MEAD offers you this threefold program



1. SOLUTIONS:

A complete line of standard and special solutions for pediatric and adult use, including the new Homeolyte Solutions, initial hydrating solutions and replacement solutions. The pediatric line includes 31 solutions, 11 exclusive with MEAD.

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New refinements include the Amifilter for protection against contamination; the Amiflo for reliable flow control; "burette-type," pediatric-size bottles graduated in 10 cc., and "memo margin" labels for dosage instructions.

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Dosage guides, calculators, conversion tables, slides, films, lecture material and new booklets on fluid therapy.

Your MEAD Parenteral Products representative can supply you with more information about the MEAD threefold parenterals program—pediatric or adult—or you may write to Parenteral Products Division, Mead Johnson & Company, Evansville, Indiana.

MEAD

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Parenteral Products Division

MEAD JOHNSON & COMPANY · EVANSVILLE, INDIANA, U.S.A.

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WHITE'S COD LIVER OIL
CONCENTRATE TABLETS
taste as good as candy. . .
kids love to chew them.
Each tablet is equivalent in its vitamin A and D content to a teaspoonful of USP cod liver oil.
Also available—economical White's Cod Liver Oil Concentrate Drops and high potency capsules.
WHITE LABORATORIES, INC.
KENILWORTH, N. J.
TASTE AS GOOD AS CANDY
KIDS LOVE TO CHEW THEM

for "This Wormy World"

SYRUP TABLETS

'ANTEPAR'*

effective against

PINWORMS
and
ROUNDWORMS

'Antepar' is well-tolerated and pleasant to take.

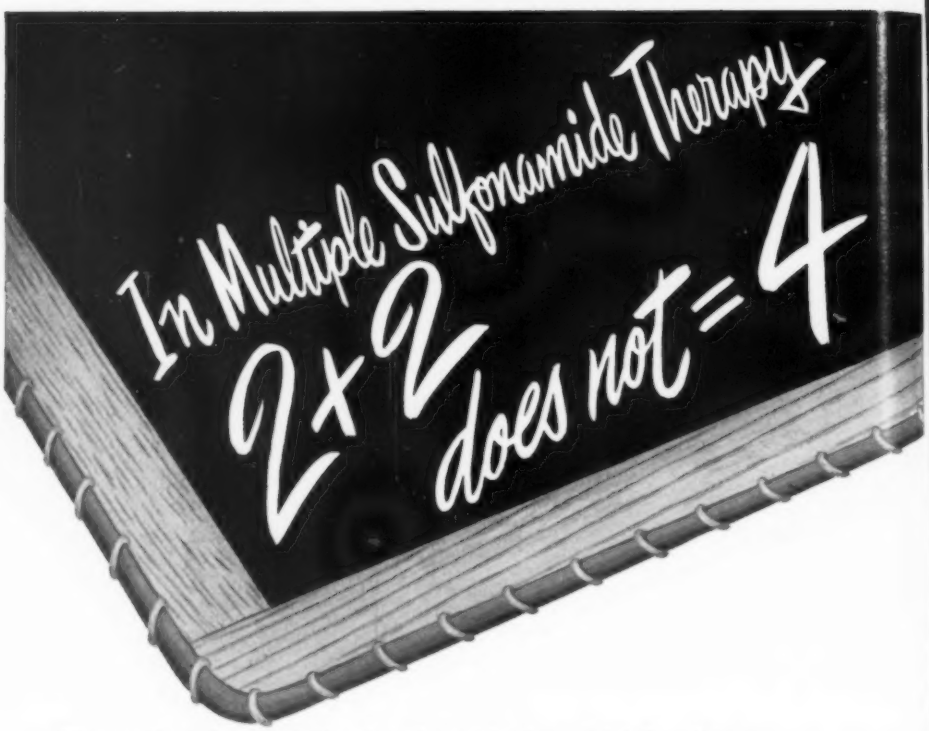
***SYRUP OF 'ANTEPAR'** Citrate brand Piperazine Citrate, containing the equivalent of 100 mg. piperazine hexahydrate per cc.

Bottles of 4 fluid ounces, 1 pint and 1 gallon.

***TABLETS OF 'ANTEPAR'** Citrate brand Piperazine Citrate, available in two strengths equivalent to either 250 mg. or 500 mg. piperazine hexahydrate, scored.

Bottles of 100

BURROUGHS WELLCOME & CO. (U.S.A.) Inc., *Tuckahoe 7, N. Y.*



Therapeutic action is additive, but the total urinary solubility of two sulfonamides is significantly greater than that of either alone.

Aldiazol-M, a dual sulfonamide, contains both sulfadiazine and sulfamerazine in microcrystalline form, and the alkalizing salt, sodium citrate. Aldiazol-M increases the field of useful application of sulfonamide therapy because:

Blood levels are produced more rapidly. The microcrystalline form of the sulfa drugs is absorbed more quickly, leading to higher initial levels.

Hazard of renal complications is reduced. Because of the greater solubility of dual sulfonamide mixture, urinary precipitation is prevented, virtually eliminating crystalluria.

Greater therapeutic efficacy is obtained. Aldiazol-M can be given safely in adequate dosage, and effective blood levels maintained in adults on a dosage of 2 teaspoonfuls every four hours.

Widely indicated. Aldiazol-M is indicated whenever the specific actions of sulfadiazine and sulfamerazine are required. It is valuable not only in adults, but also in the treatment of many infectious diseases in children.

Aldiazol-M is available on prescription through all pharmacies. Write for literature.

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Each teaspoonful (5 cc.) of Aldiazol-M provides:
Sulfadiazine (microcrystalline)...0.25 Gm.
Sulfamerazine (microcrystalline)...0.25 Gm.
Sodium Citrate..... 1.0 Gm.

Aldiazol-M

September 25. Second and third degree burns caused by flaming gasoline. Gauze pressure dressings of White's Vitamin A & D Ointment were changed at weekly intervals.

October 25. Healing is complete with minimal scar tissue and no contractures.

SEVERE BURN OR MINOR IRRITATIONS

WHITE'S VITAMIN A & D OINTMENT

Topical application of White's Vitamin A & D Ointment speeds restoration of epithelial and connective tissues. Even severe burns respond favorably to the healing action of Vitamin A & D Ointment.

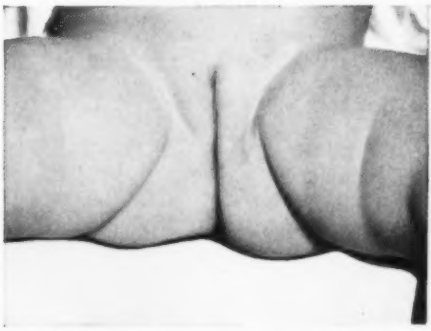
Diaper rash, chafing and abrasions also readily yield to its therapeutic and protective qualities. Prepared in suitable lanolin-petrolatum base, White's Vitamin A & D Ointment is pleasant to use, free from excessive oiliness, and will keep indefinitely. Does not stain the skin and is easily laundered from diapers or clothing.

You can prescribe it in 1½ oz. or 4 oz. tubes; 1 lb. or 5 lb. jars.

- diaper rash
- soft tissue injuries
- dry skin
- bedsores
- slow healing wounds
- varicose and diabetic ulcers
- fissured nipples

White's WHITE LABORATORIES, INC., KENILWORTH, N. J.

August 25. A typical case of diaper rash, characterized by excoriation and soreness.



September 1. After only one week of local applications with White's Vitamin A & D Ointment each time diaper was changed, the skin surface is normal.

poliomyelitis prophylaxis pitman·moore company

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...a carbohydrate of choice
in milk modification for 3 generations

OPTIMUM caloric balance—60% of caloric intake, gradually achieved in easily assimilable carbohydrates—is assured with Karo. Milk alone provides 28%, or less than half the required carbohydrate intake.

A MISCIBLE liquid, Karo is quickly dissolved, easy to use, readily available and inexpensive.

A BALANCED mixture of dextrins, maltose and dextrose, Karo is well tolerated, easily digested, gradually absorbed at spaced intervals and completely utilized.

PRECLUDES fermentation and irritation. Produces no reactions, hypoallergenic. Bacteria-free Karo is safe for feeding prematures, newborns, and infants—well and sick

LIGHT and dark Karo are interchangeable in formulas; both yield 60 calories per tablespoon.

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all through the night

one application of

DESITIN®

OINTMENT

helps protect the infant's skin against

diaper rash (ammoniacal dermatitis) • **irritation** • **excoriation**

DESITIN OINTMENT covers the infant's skin with a soothing, protective, healing coating which is largely impervious to and helps guard against irritation, rash, and maceration caused by urine, excrement, perspiration and secretions. This preventive action of Desitin Ointment *persists all through the night*...when baby is particularly vulnerable to painful skin excoriations.

Nonsensitizing, nonirritant Desitin Ointment..**rich in cod liver oil** ....successfully used on millions of infants for over 30 years.

- tubes of 1 oz., 2 oz., 4 oz.
- 1 lb. jars.

for **samples** *and literature please write....*

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1. Grayzel, H. G., Heimer, C. B., and Grayzel, R. W.: New York St. J. Med. 53:2233, 1953. **2.** Heimer, C. B., Grayzel, H. G., and Kramer, B.: Archives of Pediatrics 68:382, 1951. **3.** Behrman, H. T., Combes, F. C., Bobroff, A., and Leviticus, R.: Ind. Med. & Surgery 18:512, 1949. **4.** Turell, R.: New York St. J. Med. 50:2282, 1950. **5.** Marks, M. M.: Missouri Med. 52:187, 1955.

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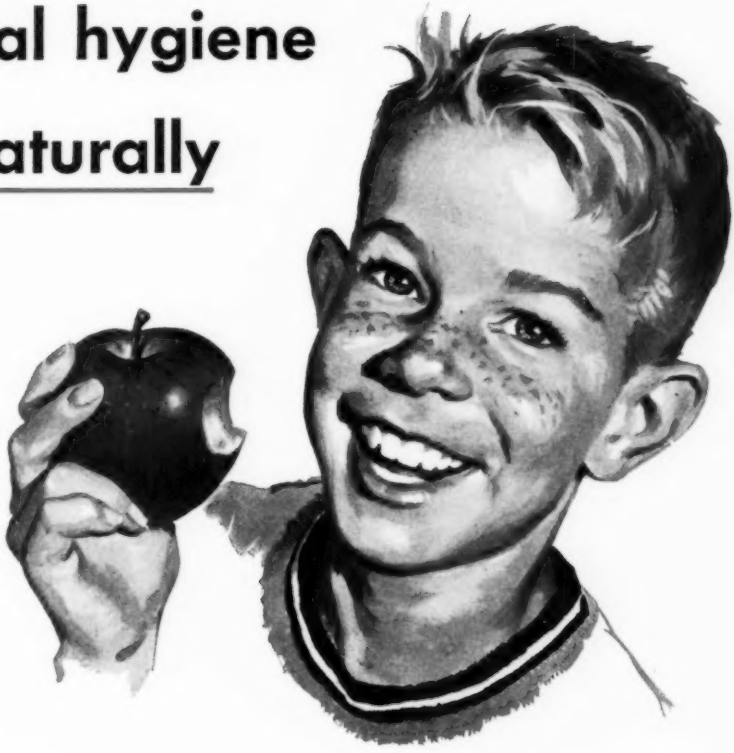
Romilar 'Roche' is at least as effective as codeine in relieving cough -- but it does not constipate and is not habit-forming. 10 mg Romilar® equals 15 mg (1/4 gr) codeine. Romilar is available as a syrup, in tablets and as an expectorant.

More and more physicians —

...are prescribing Gantrisin® 'Roche.' Why? Because they've found that this single, soluble wide-spectrum sulfonamide is usually both effective and well tolerated. There are over 300 references to Gantrisin in recent medical literature.

Apples aid dental hygiene naturally



The high incidence of tooth decay among school children gives urgency to the question: How shall mothers cope with the dangers of excessive sweets, misguided appetites, and the constant need to keep teeth clean throughout the day?

One part of the answer stands out: Apples can help. With all their familiar values, apples are a practical key to better nutritional practices for they enjoy outstanding favor with small fry. Children instinctively go for apples with their native, unspoiled taste. Sinking the teeth into a crisp, juicy apple, savoring it down to the core, the youngster benefits from a natural tooth-cleansing action.

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In Behalf of
THE APPLE GROWERS OF AMERICA

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Dropped directly on the tongue or on a spoonful of dessert or cereal, *Diatussin* lessens frequency and severity of cough. Non-narcotic, *Diatussin* preserves the vital cough reflex, avoids sedation and gastro-intestinal disturbances.

tykes don't "take on" when they take...

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non-narcotic cough control

Bischoff DIVISION

Dosage:

Under 5 years...2 to 4 drops three or four times daily. Over 5 years...5 drops three or four times daily.

Formula:	*Diatussin*
Thyme (alcoholic extract) . . .	39%
Drosera (alcoholic extract) . . .	39%
Ethyl alcohol	22%

Supplied in 6-cc. bottles with dropper.

Diatussin Syrup, in 4-oz., pint and gallon bottles, contains in each teaspoonful 2 drops of the extract in an aqueous dextrose vehicle.

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A mild castile soap specifically formulated
for babies; free from caustic alkali;
ideal for sensitive skin.

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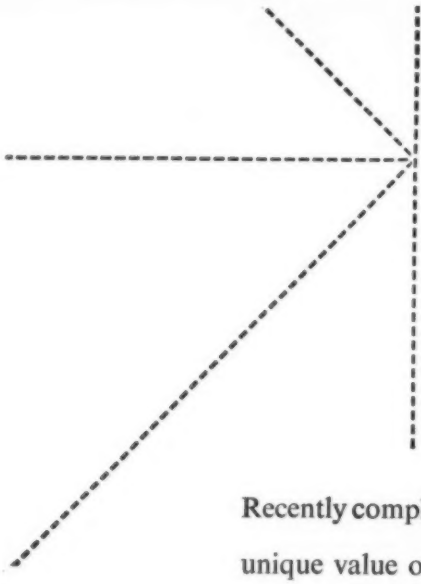

ANEMIA OF INFANCY

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Clinical results show that routine administration of Roncovite can completely prevent the iron deficiency which so frequently develops in the first six months of life.

RONCOVITE (*Cobalt-Iron*) *has introduced a wholly new concept in anti-anemia therapy. It is based upon the unique hemopoietic stimulation produced only by cobalt. The application of this new concept has led to marked, often dramatic, advances in the successful treatment of many of the anemias.*

SUPPLIED:

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Each 0.6 cc. (10 drops) provides:

Cobalt chloride.....40 mg.
(Cobalt 9.9 mg.)

Ferrous sulfate.....75 mg.

DOSAGE:

In children one year or older 0.6 cc. (10 drops); infants less than one year 0.3 cc. (5 drops); once daily diluted with water, milk, fruit or vegetable juice.

1. Coles, B. L., and James, U.: Arch. of Disease in Childhood *29*:85 (1954). Coles, B. L., and James, U.: Journal-Lancet *75*:79 (March) 1955. Coles, B. L.: Arch. Disease in Childhood *30*:121 (April) 1955.
2. Quilligan J. J., Jr.: Texas State J. Med. *50*:294 (May) 1954.

Bibliography of 192 references available on request.

EFFECTIVE

"It is a significant fact that none of the... cases receiving iron as well as cobalt required additional iron therapy and that the haemoglobin levels of this group remained consistently and significantly higher than those in any other group after the age of 4 months."[1]

"...there can be no doubt that the average hemoglobin values... are greater in the cobalt-iron (Roncovite) treated group."[2]

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"...the mothers of these anaemic infants frequently stated spontaneously that the children were much improved, with increased appetite and vigour. It seems possible, therefore, that even if anaemia in premature infants does not usually produce marked symptoms, there is a subclinical debility which becomes more evident in retrospect."[1]

SAFETY

"There was no evidence of toxicity in any case under treatment:...There is nothing to suggest that cobalt in any way impairs the general progress or rate of weight gain in premature infants in the dosage employed."[1]

"The babies were closely observed daily for ill effects of the medication while at the premature unit and when they returned for check ups. None of them showed harmful effects despite the large doses... A few of the babies ...have been followed for more than 100 days with no ill effects noted."[2]

RONCOVITE

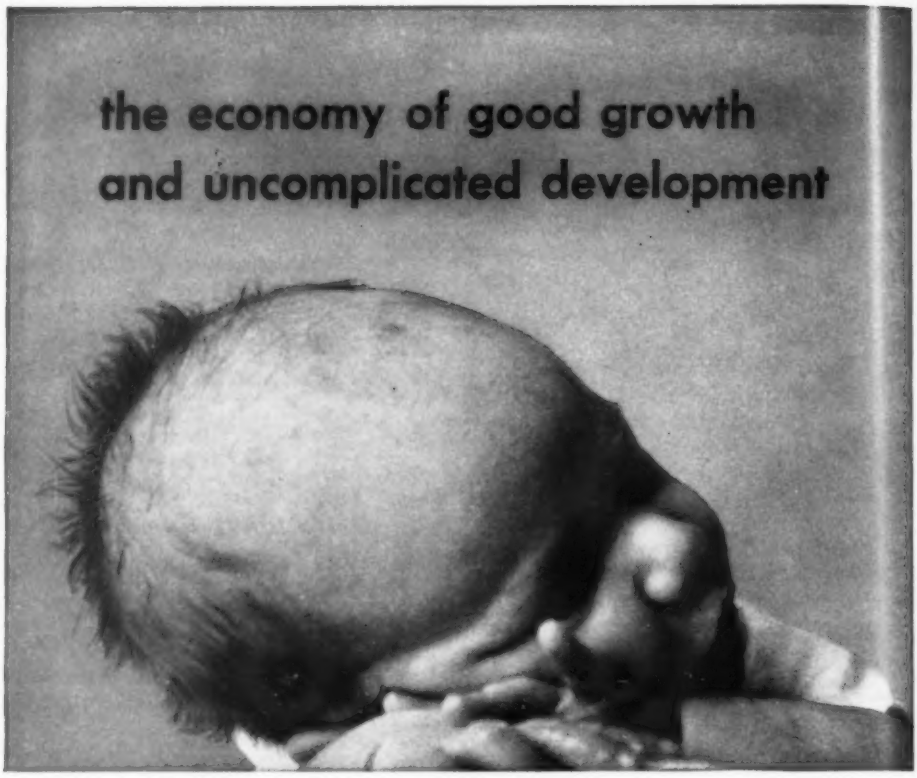
The original, clinically proved cobalt-iron product.

LLOYD

BROTHERS, INC.

Cincinnati, Ohio

In the Service of Medicine Since 1870



SIMILAC POWDER—*physiologic food during the first year of life*—

To assure sound growth and reduce many of the complications commonly encountered in the first year of life, the full, balanced Similac formula provides: fat, protein and carbohydrate closely approximating the content of human breast milk in quality and quantity; a full complement of known essential vitamins in adequate amounts; an adjusted mineral content; a soft, fluid curd with zero tension, assuring rapid and easy digestion.

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SIMILAC *powder*

There is no closer equivalent to the milk of healthy, well-nourished mothers

SUPPLIED:
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M & R LABORATORIES, *Columbus 16, Ohio*

a complete B complex formula derived from pure beef liver

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Niacinamide ... 10 mg.	Inositol ... 10 mg.
Pyridoxine HCl (B_6) ... 0.2 mg.	Soluble Liver Fraction ... 470 mg.
Pantothenic Acid ... 2 mg.	Vitamin B_{12} ... 5 micrograms

LEDERLE LABORATORIES DIVISION *AMERICAN Cyanamid COMPANY* Pearl River, New York

LEDERPLEX*

Vitamin B Complex LIQUID *Lederle*

Lederle also offers LEDERPLEX in Tablet, Capsule, and Parenteral form.

*REG. U.S. PAT. OFF.

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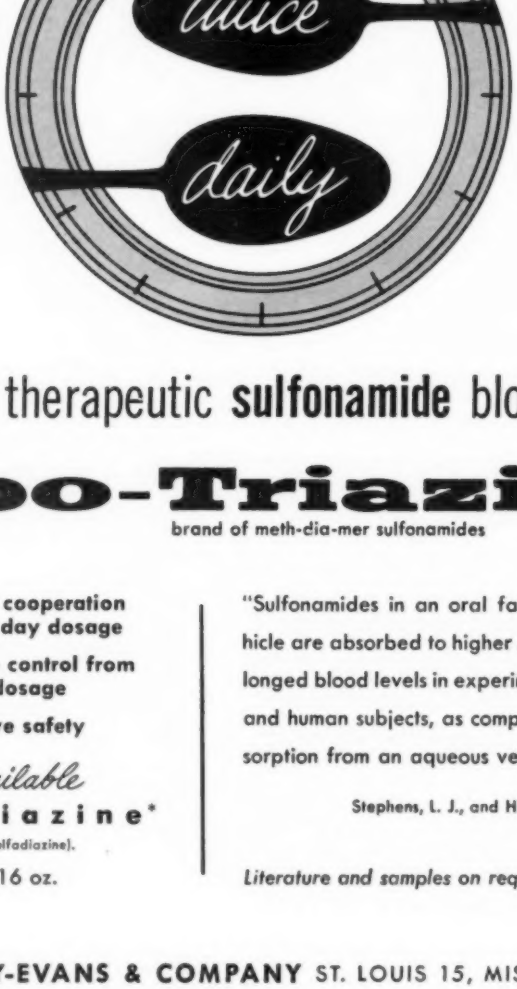

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REFERENCES: (1) Eigel, E.: Personal communication. (2) Stephens, LeR. J.: Personal communication. (3) Hiatt, R. B., and Wilson, P. E.: Surg., Gynec. & Obst. 87: 317, 1948. (4) Andersen, D. H.: Pediatrics 3: 406, 1949; Personal communication, 1948. (5) Andersen, D. H., and di Sant' Agnese, P. A.: True or Idiopathic Celiac Disease, in Brennemann: Practice of Pediatrics, Hagerstown, Md., W. F. Prior Co., vol. 1, chapter 29, 1952. (6) Vignec, A. J.: Personal communication, 1952.

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PLUS essential iron and calcium.

2 measures (2.3 Gm.) of Lactofort supply:

L-lysine (from L-lysine monohydrochloride)	500	mg.
Vitamin A acetate	3750	U.S.P. Units
Vitamin D	1000	U.S.P. Units
Thiamine mononitrate	0.75	mg.
Riboflavin	1.25	mg.
Niacinamide	7.5	mg.
Vitamin B_{12}	2.5	mcg.
Folic Acid	0.25	mg.
Ascorbic acid (from sodium ascorbate)	75	mg.
Pyridoxine hydrochloride	0.75	mg.
Calcium pantothenate	7.5	mg.
Iron ammonium citrate green (elemental iron 7.5 mg.)	50	mg.
Calcium gluconate (elemental calcium 130 mg.)	1.45	Gm.

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Meat...

and the Value of Fat in Nutrition

Authorities in the field of nutrition no longer consider fat as an optional component of the diet. Evidence from the laboratory and bedside indicates that fat in small amounts may be looked upon as an obligatory constituent of a health-promoting diet.[1]

The far-reaching value of fat in nutrition has been amply demonstrated in laboratory animals in its pronounced effect on growth, on pregnancy and lactation, on nitrogen-sparing action, on work capacity, on time of sexual maturity, on the period of survival during fasting, and on ability to combat external stresses.[1]

Young animals fed a fat-free diet not only fail to grow normally, but develop hair and skin changes characteristic of "essential" fatty acid deficiency.[2] Fatty acids other than the "essential" fatty acids also appear to be necessary for optimal health. Animals fed "essential" fatty acids but no others do not grow optimally.

The value of fat in human nutrition was emphasized in a recent study[2] comprising 200 patients incapable of receiving adequate nourishment. For periods of 1 to 30 days, these patients were given supplementary fat alimentation by vein in the form of fat emulsion containing "essential" as well as other fatty acids. The result was typically a marked increase in weight and more positive nitrogen and potassium balances.

Meat, recognized for its high content of biologically valuable protein, B vitamins, and essential minerals, provides, in addition, substantial amounts of nutritionally important fat.

1. Deuel, H. J., Jr.: Newer Concepts of the Role of Fats and of the Essential Fatty Acids in the Diet, Food Res. 20:81 (Jan.-Feb.) 1955.

2. Meng, H.C.: Preparation, Utilization, and Importance of Neutral Fat Emulsion in Intravenous Alimentation, in Najjar, V.A.: Fat Metabolism, Baltimore, The Johns Hopkins Press, 1954, pp. 69-92.

The nutritional statements made in this advertisement have been reviewed and found consistent with current medical opinion by the Council on Foods and Nutrition of the American Medical Association.

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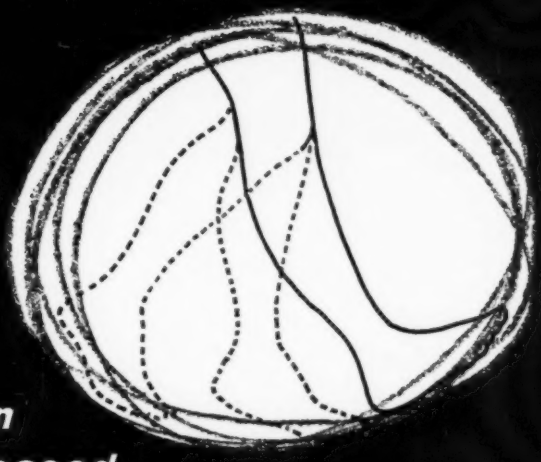

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preliminary findings,[1] based on the measuring of pituitary ACTH suppression potency of various corticoids, appear to indicate that STERANE is 20% more potent than the cortisone analog, prednisone

1. Forsham, P. H., et al.: Paper presented at First Internat. Conf. on Prednisone and Prednisolone, New York, N. Y., May 31-June 1, 1955.

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Bibliography: (1) Henderson, E.: New developments in steroid therapy of rheumatic diseases, presented at New Jersey State Medical Society Meeting, Atlantic City, New Jersey, April 17-20, 1955. (2) Clinical Research Division, Schering Corporation: Compilation of investigative reports. (3) Criep, L. H.: Prednisolone and prednisone in the treatment of allergic diseases, to be published. (4) Robinson, H. M., Jr.: First International Conference on Meticorten and Meticortelone, New York City, May 31-June 1, 1955. (5) Sternberg, T. H., and Newcomer, V. D.: Am. Pract. & Digest Treat. *6*:1102, 1955. (6) Bunim, J. J.; Pechet, M. M., and Bollet, A. J.: J.A.M.A. *157*:311, 1955. (7) Waine, H.: Bull. Rheumat. Dis. *5*:81, 1955. (8) Tolksdorf, S., and Perlman, P.: Fed. Proc. *14*:377, 1955. (9) Herzog, H. L., and others: Science *121*:176, 1955. (10) Bunim, J. J.; Black, R. L.; Bollet, A. J., and Pechet, M. M.: Ann. New York Acad. Sc. *61*:358, 1955. (11) Boland, E. W.: California Med. *82*:65, 1955; abs., Curr. M. Digest *22*:53, 1955. (12) King, J. H., and Weimer, J. R.: A.M.A. Arch. Ophth. *54*:46, 1955. (13) Gordon, D. M.: Prednisone and prednisolone in ocular disease, to be published.

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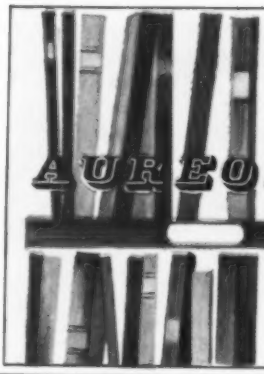

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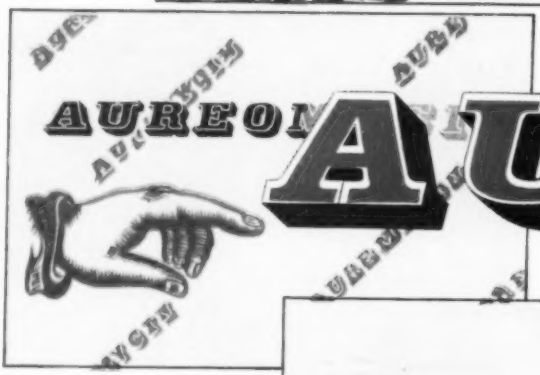

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1. Porter, L., and Carter, W. E.: Management of Sick Infant and Child. St. Louis, C. V. Mosby Co., 1942, Ed. 6, p. 93.

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1. Shackman, N. H.: Heffer, E. T., and Kroop, I. G.: Am. Heart J. 48:599 (Oct.) 1954. • 2. Stollerman, G. H., and others: Am. J. Med. 15:645 (Nov.) 1953. • 3. McEwen, C.: M. Clin. North America 39:353 (March) 1955. • 4. Wood, H. F., and McCarty, M.: Am. J. Med. 17:768 (Dec.) 1954. • 5. McEwen, C., and Ziff, M.: M. Clin. North America (May) 1955, to be published.

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Pennsylvania M. J., 58:383 (April) 1955.

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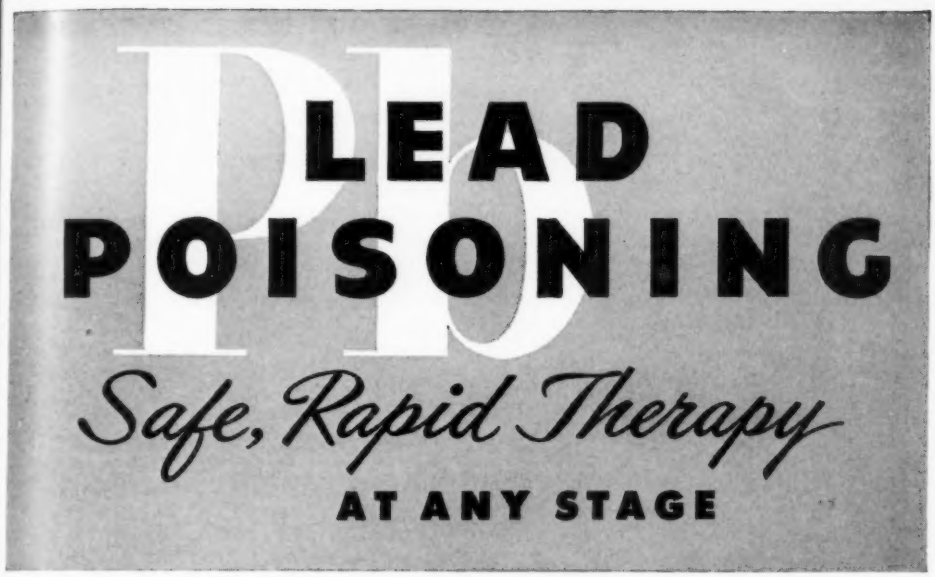

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1. Rubin, M.: Biological Action of Versene, from Symposium on Complexing Agents, presented at the University of Chicago, Feb. 22, 1951.
2. Sidbury, J. B., Jr.; Bynum, J. C., and Fetz, L. L.: Effect of Chelating Agent on Urinary Lead Excretion. Comparison of Oral and Intravenous Administration, Proc. Soc. Exper. Biol. & Med. 82:226 (Feb.) 1953.
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4. Bradley, J. E., and Powell, A. M., Jr.: Oral Calcium EDTA in Lead Intoxication of Children, J. Pediat. 45:297 (Sept.) 1954.

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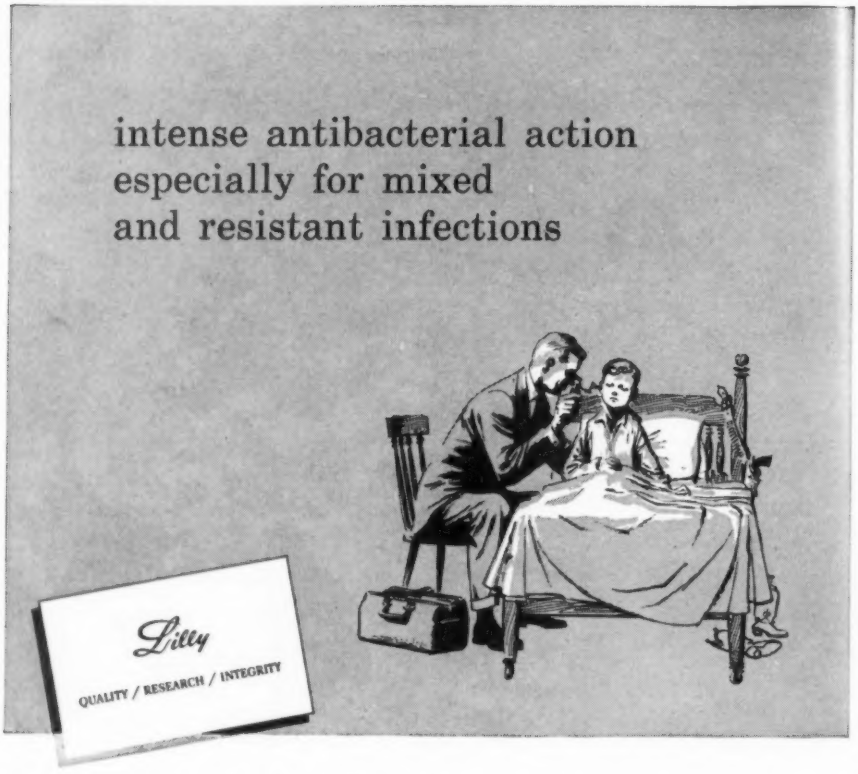

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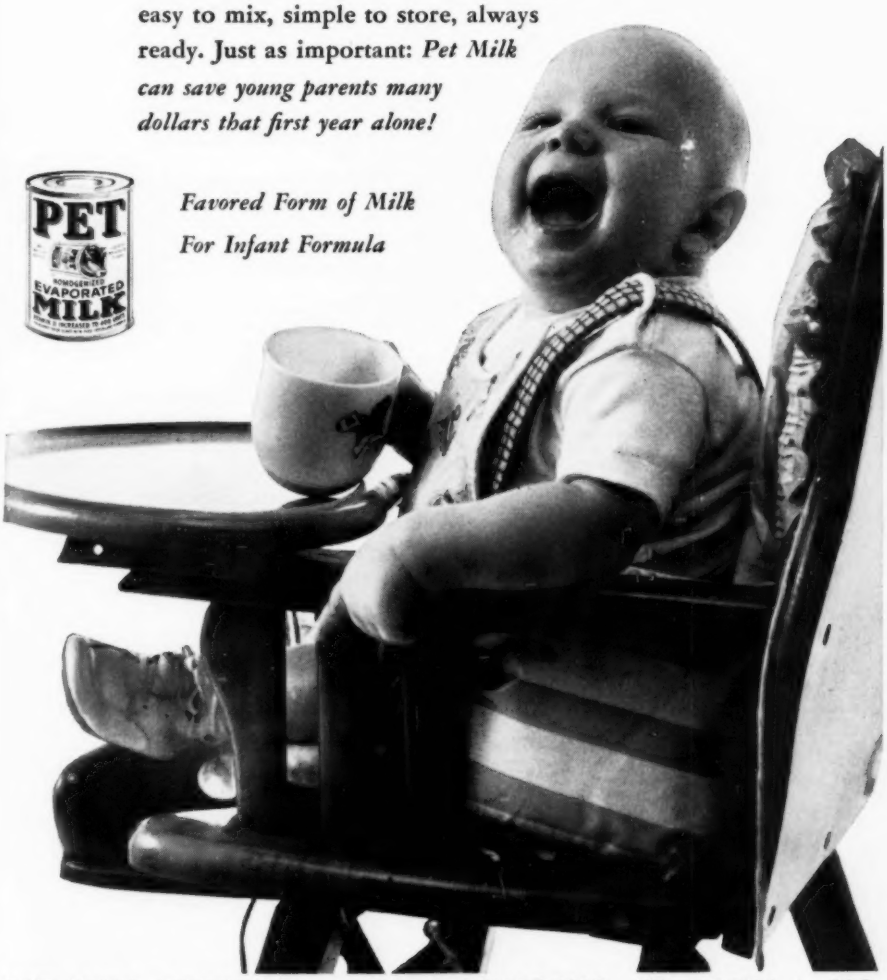

PET MILK COMPANY, ARCADE BUILDING, ST. LOUIS 1, MO.

Systemic Associations and Treatment

of

SKIN DISEASES

By KURT WIENER, M.D.
Dermatologist, Mount Sinai Hospital, Deaconess Hospital,
Saint Michael's Hospital, Milwaukee, Wisconsin

556 Pages, 90 Illustrative Plates

PRICE, $17.00

The author's earlier work entitled "Skin Manifestations of Internal Disorders" covered the skin changes which accompany internal or systemic diseases.

This new book looks in the opposite direction. It is concerned with the systemic phenomena associated with skin diseases.

The systemic, i.e., extracutaneous, relations of all skin diseases of importance such as eczema, psoriasis, urticaria, pemphigus, lupus erythematosus and many others are fully discussed.

The chapters on Systemic Therapy of skin diseases represent a third aspect of the complex which one might call systemic dermatology. Systemic therapy includes not only the nontopical drugs, but also diets, indirect radiation therapy and balneologic methods. Some light is also thrown on unorthodox methods.

The book is divided into two parts —Part One covering Systemic Associations of Skin Diseases; Part Two being devoted to Systemic Treatment of Skin Diseases. To a large degree the same pattern is followed in the discussion of each disease or condition. For example: In Part One the Incidence is first taken up, followed by the Systemic Relationship and Association, then Systemic Manifestations, Systemic Treatment. Frequently the Clinical Picture is given, along with the Prognosis, Pathology, Etiologic Factors, Diagnosis, Laboratory Findings—and, of course, details of Treatment. In Part Two the pattern is to discuss the Chemistry and Physiology, Clinical Application, Administration and Dosage, Side Effects, Results and Indications, Mode of Action, Toxicity, Precautions, Pharmacology and Contraindications.

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Date _____

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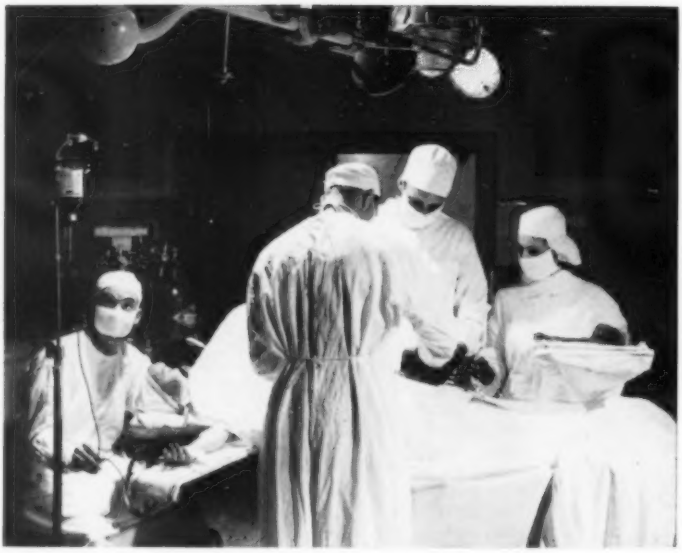
Red Cross Blood Program



YOU know when blood is needed it's needed immediately and often in large quantities . . .

YOU know when sickness or accident or disaster hits an individual or family or community, it's seldom announced in advance—it strikes quickly, suddenly.

YOU know when the chips are down, people generally help in whatever way they can.

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Blood must be donated before the need . . . a safety measure.

Every 20 seconds in 1953 some hospital patient in the United States received a blood transfusion. If the nation's civilian ill and injured are to continue getting the blood they need, 1 out of 20 persons 18 through 59 years of age will have to make at least one blood donation a year.

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YOU can encourage people to make a blood donation once or twice a year through the Red Cross Blood Program, the community nonprofit blood bank, or the hospital in your community.

YOU can help people help you save lives!

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Penicillin V, Crystalline (Phenoxymethyl Penicillin)

the totally new penicillin for decisive oral dependability

Supplied: Tablets, 125 mg. (200,000 units), bottles of 36. Also available: Tablets BICILLIN®-VEE, 100 mg. (100,000 units) of benzathine penicillin G and 62.5 mg. (100,000 units) of penicillin V, bottles of 36.

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In the control of allergic symptoms a combination of three antihistamines means greater safety.

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an effective, safer combination of three antihistamines.

Available in Capsules, and exceptionally palatable fruit-flavored Syrup (half-strength) for children.
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Select the level of protection the baby needs

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10 Nutritionally Significant Vitamins
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TARBONIS with HYDROCORTISONE

Each 0.6 cc. supplies:

	Vitamin B_{12} (mcg.)	Biotin (mcg.)	Panthenol (mg.)	Pyridoxine (mg.)	Nicotinamide (mg.)	Riboflavin (mg.)	Thiamine (mg.)	Ascorbic acid (mg.)	Vitamin D (units)	Vitamin A (units)
POLY-VI-SOL 6 Essential Vitamins	1	30	3	1	10	1.5	1	50	1000	5000
TRI-VI-SOL 3 Basic Vitamins					6	0.8	1	50	1000	5000

All are supplied in 15 cc., 30 cc. and economical 50 cc. bottles with the new Mead calibrated unbreakable plastic 'Safti-Dropper.' It will not break even if the baby bites it.

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MEAD JOHNSON & COMPANY • EVANSVILLE, INDIANA, U. S. A.

REFRACTORY SUB-ACUTE AND CHRONIC DERMATOSES RESPOND TO TARCORTIN

SYNERGISTIC COMBINATION OF TARBONIS AND HYDROCORTISONE

EFFECTIVE WHERE OTHER THERAPY FAILED

WHAT IT IS: TARCORTIN is a synergistic combination of 0.5% hydrocortisone in Tarbonis (a greaseless, stainless, vanishing cream containing 5% of a special extract of coal tar)

WHAT IT DOES: TARCORTIN is effective in simple and refractory sub-acute and chronic dermatoses. The synergistic action brings better results with lower doses of hydrocortisone, and irritation is not encountered even in intertriginous areas

WHAT IT IS FOR: TARCORTIN is indicated in the treatment of all sub-acute and chronic dermatoses - localized neurodermatitis chronic eczema, hand eczema, seborrhea, atopic eczema, dermatitis venenata, nummular eczema, pruritus ani and vulvae, psoriasis, etc.

CLINICAL PROOF: 100 patients, suffering from sub-acute and chronic dermatoses, used TARCORTIN for an average of three weeks. The name of the product and its composition was unknown to the patients to rule out improvement on any psychological basis. 95% of the cases improved.

"In all cases . . . the combination provided prompt and marked relief. The results compared favorably with cases in which twice the amount of hydrocortisone was used without tar" [1]

AVAILABLE: TARCORTIN—¼ ounce tubes

1—Literature available

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"JUNKET" Rennet-Custard . . . the fresh-milk dessert . . . is among the first solid foods in the infant dietary. Served with interesting variations, it is a light, wholesome dessert . . . good for the whole family.

Is there a difference in "Baby Desserts"?

the answer is below

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	Cal.	Prot. gm.	Fat gm.	Carbo. gm.	Ca. mg.	P. mg.	Vit. A I.U.	Ribo. mg.
*DESSERT A	94	2.0	2.7	20.2	76	63	Trace	0.111
*DESSERT B	100	2.32	1.89	17.72	76	43	25 (USP)	0.160
*DESSERT C	102	2.4	3.2	16.5	75	69	228	0.110
**RENNET-CUSTARDS (average of seven flavors)	96.5	3.2	3.6	12.7	123	102	175	0.164

*Nationally distributed "ready-to-serve" desserts.
**Made with fresh milk and "Junket" Rennet Powder.

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Makes *Fresh* Milk into Rennet-Custards

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"Junket" Rennet Tablets: Not sweetened or flavored.

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IN CORTICOID-TREATED PATIENTS

minimize adrenal suppression and atrophy

BY THE REGULAR PERIODIC USE OF

HP*ACTHAR® *Gel*

Stress of surgery, accidents or infections is magnified in patients treated with cortisone, hydrocortisone, prednisone or prednisolone. Adrenal steroids, even in small doses, jeopardize the defense mechanism against stress by causing adrenal cortical atrophy. Concomitant use of HP*ACTHAR *Gel* counteracts adrenal atrophy by its stimulant action on the adrenal cortex.

Dosage recommendations for supportive HP*ACTHAR *Gel* are, inject:

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b. 100 U. of HP*ACTHAR *Gel* for every 200 to 300 mg. of hydrocortisone.

c. 100 U. of HP*ACTHAR *Gel* for every 400 mg. of cortisone.

2 Discontinue use of steroid on the day of injection.

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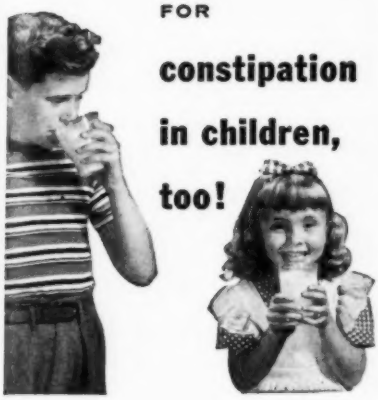
FOR

constipation in children, too!



IN MILK...
tastes like malted milk

Dose: 2 tablespoonfuls Malt Soup Extract in a glass of warm milk at bedtime. It may take 2-3 days to soften stools. Then 1-2 tablespoonfuls in warm milk at bedtime to maintain softness.

Borcherdt

MALT SOUP Extract*

Pediatricians like Malt Soup Extract *for children of all ages* because it provides a laxative beverage that softens dry, hard stools . . . is not habit-forming.

Children like Malt Soup Extract in milk because it has a delicious "malted" taste that's a real treat.

Mothers like Malt Soup Extract because youngsters welcome it without coaxing.

*Specially processed malt extract neutralized with potassium carbonate. In 8 oz. and 16 oz. bottles.

Samples and literature on request

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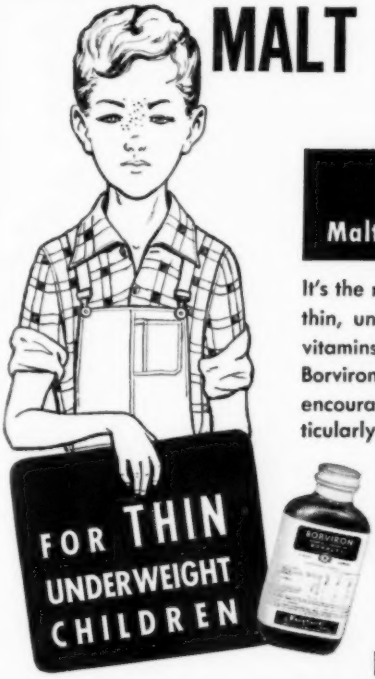
WEIGHT GAIN . . . CONVALESCENCE



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BORVIRON
Malt - Iron - Vitamin Supplement for Children

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the baby needs

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10 Nutritionally Significant Vitamin
INCLUDING VITAMINS B_{12} AND B_6

Deca-Vi-Sol—the new, more comprehensive formula including vitamins B_{12} and B_6—permits even greater flexibility in specifying vitamins for infants and children. Like Poly-Vi-Sol and Tri-Vi-Sol . . .

Deca-Vi-Sol is **highly stable** . . . refrigeration not required . . . potency assured . . . **readily accepted** . . . exceptionally pleasant flavor . . . no unpleasant aftertaste . . . **full dosage assured** . . . can be dropped directly into the baby's mouth.

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. . . Rapid, intense and prolonged analgesic action with the complemental anesthetics, zolamine and Eucupin.®
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	1	30	3	1	10	1.5	1	50	1000	5000
POLY-VI-SOL 6 Essential Vitamins					6	0.8	1	50	1000	5000
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Broad Antibacterial, Antifungal Activity in External and Chronic Middle Ear Infections

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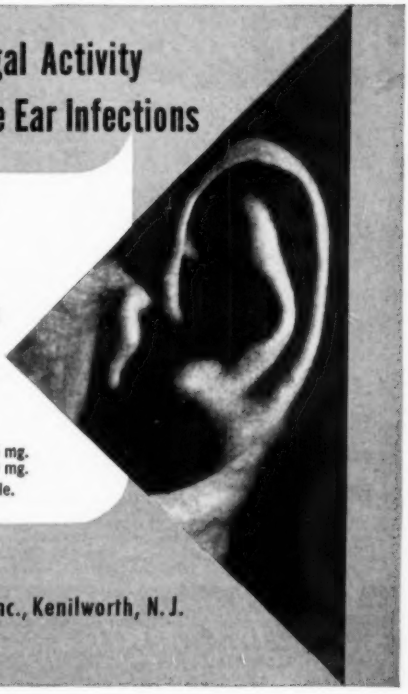

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Neomycin Sulfate (equivalent to neomycin base) . . . . . 3.5 mg.
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... in an autogenously sterile hydroalcoholic glycerin vehicle.

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tastes just like

banana-flavored

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New Ready-Mixed Penicillin Suspension

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don't gamble . . . *with baby's tender skin!*

make your point, doctor . . . *prescribe* . . .

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cleanses tender skin gently . . . *without irritation*

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Supplied as convenient 4 oz. bars

Soapless but lathers copiously . . . contains no alkali or other irritating components of soap. Its lather is so mild . . . *does not make baby's eyes smart.* Preserves the protective "acid mantle" of the skin.

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1. Devlin, L. P.: Enteritis in Industrial Medicine—Carob Flour (Arobon) in Therapy, Indust. Med. & Surg. 23:166 (Apr.) 1954.

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OFTEN HELP CORRECT CHILDHOOD CONSTIPATION

In learning bowel control, some children "overtrain," developing tight rectal muscles which results in constipation. Others become so busy they delay defecation, which again may cause abnormal tightness of the sphincture. Dilatation stretches rectal muscles toward normal. Use of Young's dilators for several days often re-establishes evacuation "habit time" without laxatives.

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When small patients begin to burn, the time is right for a spoonful or two of elixir

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Tylenol brings the fever down promptly, eves aches, pains and other febrile miseries. ylenol is just about twice as safe as aspirin. Its taste is readily acceptable to children. Used one or in conjunction with specific therapy. ideal choice, indeed, for pediatric antipyresis.

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Select the level of protection the baby needs

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	1	30	3	1	10	1.5	1	50	1000	5000
POLY-VI-SOL 6 Essential Vitamins					6	0.8	1	50	1000	5000
TRI-VI-SOL 3 Basic Vitamins								50	1000	5000

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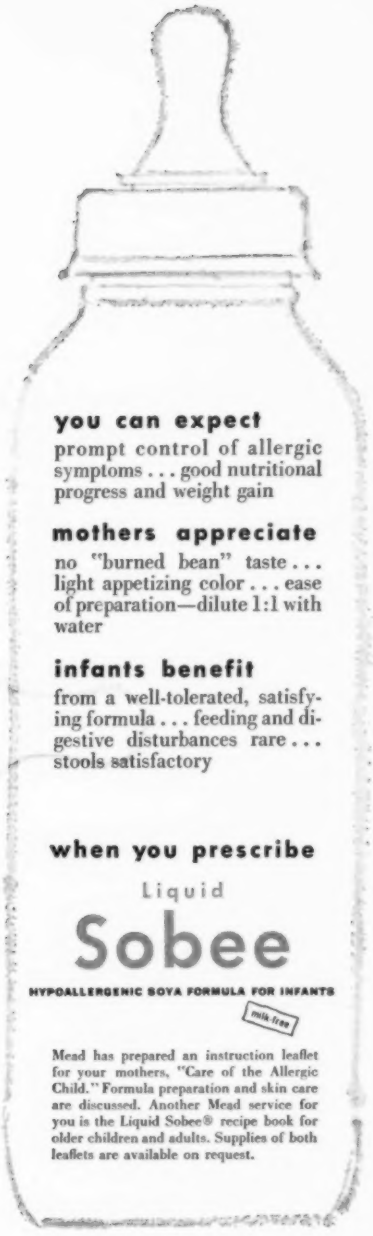

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1. Ratner, B. et al: A.M.A. Am. J. Dis. Child. 89:187, 1955.
2. Glaser, J., and Johnstone, D. E.: J.A.M.A. 153:620, 1953.

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